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NOTES ON DISRUPTION

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There is a curious reticence on the subject of disruption of abdominal wounds. Most surgeons are ready at the drop of a hat to record the number of gastrectomies they have perpetrated. They are equally ready to describe solitary examples of rare cases in papers of great length and greater boredom. But it looks as if familiarity with burst abdominal wounds carries a stigma. Good surgeons do not see disruptions. True or false?

A recent report (Tweedie and Long, 1954) from the Royal Victoria Hospital at Montreal reveals an incidence of 1 in 200 abdominal and gynaecological operations in a total of over 22,000. Despite advances in anaesthesia and in the use of antibiotics and physiotherapy the number of cases is slightly on the increase. If in addition we look upon incisional herniae as partial disruptions, which indeed most of them are, we see that this is a subject which warrants discussion and is of great practical importance.

The complication is always annoying, almost always unexpected, and not infrequently calamitous. I can recall only one case (years ago) where the bursting open of an abdominal wound was welcomed. It followed a cholecystectomy on a plethoric butcher. The cystic-artery ligature had come off in the struggle. Finally, when my blood pressure was in inverse proportion to that of the patient and the abdomen was closed we were told that a swab was missing. Sure that all was well I did not re-open. But the missing swab was never found. A week later the patient obligingly coughed open his wound. I was able to have a 'second look' and to make quite certain that it had not been left in the abdomen.

THE CAUSES OF DISRUPTION

How does disruption come about? The simple facts of wound healing are familiar enough to most surgeons. For about 4 or 5 days after the wound is made there is a lag in the reparative process. During this time the integrity of the wound depends upon the tensile strength of the suture material and the tissue it approximates. At the end of this period fibroblasts appear and secrete the collagen which will constitute the scar. The latter stage is progressive, i.e. in time the argentophil fibrils (pre-collagen) tend to become disposed along the lines of mechanical stress; they grow shorter and stronger and the eventual scar becomes resistent to strain.

A thyroid incision closed with Michel clips will not disrupt even if the clips are removed in 24 hours; similarly with a hernia incision. It is obvious that the unique feature of disruption in abdominal wounds is the varying intra-abdominal tension to which they are subjected. From this observation we may answer most of the questions which follow logically from the main one: how does disruption arise?

Suture Material

Is it due to defective suture material? Yes, sometimes. A frayed continuous catgut suture may yield under stress. But steel wire may do the same. My most recent case occurred after an abdomino-perineal resection of the rectum for cancer, where the wound burst open after 2 days and where the cause was plainly a broken continuous steel suture. In this connection we must ask ourselves which layer is important in the security of the wound. Most surgeons use catgut for the suture of peritoneum. If the peritoneal layer yields and the muscular and fascial layers hold will the wound burst open? Other factors remaining equal (absence of distension, no coughing, no straining at stool) the answer is probably not—for peritoneum has an amazing regenerative power. On the other hand if the peritoneal layer holds and the fascial layer gives way the wound will almost certainly burst open. The important layer is therefore the fascial, and during the lag period the suture material uniting this layer is of great importance. Of course if it is defective it will give trouble whatever its nature, but it is well to remember that, no matter how

strong the suture, if the fascia or peritoneum it approximates is friable or flabby the wound will open. But on the whole it is better to use non-absorbable continuous wire or nylon for vertical or transverse incisions in preference to catgut. The McBurney incision does not require this; disruption here is unknown.

Careless Suturing

Is dehiscence caused by careless suturing? Yes, quite often. Leaving a bit of omentum between two bites of peritoneum is asking for trouble. Insecurely tied knots and tears in a thin peritoneum favour disruption. Closure of the wound at the end of a long operation is the ha'porth of tar for lack of which the whole ship may founder. If the surgeon is tired he should allow his assistant to sew up.

Defective Healing

Is a defect in healing power responsible for disruptions? Yes, sometimes. Vitamin C has been experimentally shown to be essential in the healing of wounds and although this factor is not so important in humans it cannot be ignored. A high-protein diet hastens healing. Therefore, in patients who are poorly nourished either through their disease or their circumstances, disruption may occur in the stage where the maturation of their fibroblasts and the deposition of collagen should be most active, i.e. after the 6th day. Hypoproteinaemia was probably responsible for one of my cases on the 12th day after procto-colectomy for ulcerative colitis.

Age

Is the age of the patient a factor? Whilst wounds may open in infants and young children this occurs more frequently in the aged, where healing is slow, and particularly where the case is one of cancer. In 2 of my cases one followed hemicolectomy in an old lady of 82 and the other abdomino-perineal resection in a man of 76. On the other hand colectomy in a man of 91 left a soundly healed scar.

Intra-abdominal Pressure

What part does increase in intra-abdominal pressure play? This is probably the most important causative agent. We have seen that this is what distinguishes abdominal wounds from those in other regions. Anything which causes distension and increase in pressure will produce strain on sutures. Coughing or straining at stool or ileus are potent causes. In one of my cases, where colectomy had been done for extensive diverticulitis with mild peri-colic suppuration, I was unwise enough to perform a one-stage resection. Convalescence was greatly prolonged by the need to re-suture the wound after disruption. If I had made a temporary colostomy or caecostomy this might have been avoided. A word must be added on coughing. Patients should be encouraged to cough; good physiotherapists insist on it in the 'stir-up' after operation. But the wound must be supported and the legs bent at the knees. Either the nurse or the patient must hold a firm hand over the wound during the cough. One of my patients burst his wound straining in the lavatory on the 12th day after operation.

I am beginning to wonder whether we should go back to the abdominal binder and the many-tailed bandage with firm pressure on the wound till the stitches are removed. They were discarded because they were held to embarrass respiration. But since deep breathing, coughing and early ambulation are routine practice the danger is not so great as it was when patients lay like logs for 12 or 14 days.

Early ambulation does *not* increase the tendency to disruption. Manometric recordings with the patient up and about show no marked increase in intra-abdominal pressure. Nor is sepsis an important factor, for the use of antibiotics has not diminished the incidence.

Thus we may say that no one cause will explain why an abdominal wound bursts open. Disruptions are like aeroplane disasters; as long as the human factor is present the occasional case will go wrong. While it is true to say that the good surgeon should see very few cases it is also true that a small percentage will be found in the records of most surgeons who do major abdominal surgery. There is only one way to limit the number in one's own practice, and that is always to be fearful of it.

MORTALITY RATE

Once a wound has to be re-sutured the mortality rate for the particular operation increases. Two out of my 8 cases over the past 6 years died. The first was a patient of 82 with an extensive carcinoma of the caecum, who succumbed after re-suture. The second was a patient who burst his wound 12 days after removal of the whole colon and rectum for ulcerative colitis. He died from a coronary thrombosis after re-suture. Figures from other surgeons vary from 15% to 25%. It is true that patients sometimes die after disruption from the original disease rather than the complication, but often it occurs in just those cases where one is proudest of one's work and most hopeful of success.

It is strange how rare it is for a wound to disrupt more than once. One case in my wards at Groote Schuur (not my own) had to be re-sutured 3 times but I think this is a unique occurrence. The reason usually given for the rarity of multiple disruptions is that the lag period does not begin anew, i.e. fibroplasia goes on uninterruptedly. The explanation is not entirely satisfactory, for presumably the original cause is still present and the new scar tissue must take several days to become firm. Probably the interrupted through-and-through sutures usually employed in re-suture are strong enough to hold the edges of the wound together. Incisional hernia is however a common sequel; so we must assume that partial disruption does in fact take place. It is tempting to conclude that through-and-through non-absorbable sutures should be used when the complication is anticipated. It is however a clumsy way of closing an abdomen and moreover is not without risks of its own.

THE PALE-PINK MENACE

If after operation a pale-pink serous discharge is noted on the dressing take the patient to the theatre at once and re-open the wound. A policy of 'wait and see' ('perhaps it will hold') is simply delaying the inevitable. Strapping the edges with elastoplast is futile.

In some cases the skin is intact and the protruding viscera form a visible swelling. At this stage the trouble may not be spotted. It may be mistaken for a haematoma or abscess. But the tell-tale pink discharge will point to the diagnosis. At an early stage the disruption may be minimal and if re-suture is done before coils of bowel have prolapsed through the wound the mortality rate will be lower and a more careful closure may be possible.

Rupture of the wound is a complication of abdominal surgery which tends to be forgotten but its incidence is a definite addition to the morbidity and mortality of this branch of surgery.

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POLIOMYELITIS VACCINE: STATEMENT BY MINISTER OF HEALTH: POLIOMIËLITIS-ENTSTOF: VERKLARING DEUR DIE MINISTER VAN GESONDHEID

The following statement was issued by the Minister of Health, the Hon. J. F. Naudé on 4 November 1955:

Two batches of poliomyelitis vaccine prepared by the South African Poliomyelitis Research Foundation Laboratories were available for issue this year. Both of these batches had successfully passed the safety tests required by the latest regulations of the United States Public Health Service.

Because of the accidents with poliomyelitis vaccine that had been reported in America, the Committee of Experts called by the Minister to advise him on the release of the South African vaccine recommended that the full safety tests should be repeated on every batch prior to its issue in order doubly to ensure the safety of the vaccine.

The first batch of vaccine, which then had not been bottled, again successfully passed these tests and was issued for use in September and October. No untoward reactions worth mentioning have been reported in the numerous children inoculated with this vaccine.

The second batch of vaccine, which would only have been required a month after the issue of the first batch, had been bottled and a chemical preservative added. This chemical preservative was found to interfere with the carrying out of the safety tests and, because of this technical difficulty, it has not been possible to complete these repeat safety tests to date. This difficulty may be overcome but a further month will elapse before the results of these further tests will be available. As this will then be well into the summer months, when poliomyelitis is generally most prevalent, the likelihood of coincidental cases of poliomyelitis following vaccination will be greater than at other times. The Poliomyelitis Research Foundation has advised the Department of Health accordingly and the Minister has decided that under the circumstances it would not be wise to issue this batch of vaccine this year.

Further issues of the vaccine will consequently not be made until next winter, when the probability of the occurrence of coincidental cases will be at its lowest.

In the meanwhile the public may be assured that the single inoculations given to date, as indicated by recent American experience, should confer a significant degree of protection and also that the delay of several months in giving the second inoculation will not affect the development of a subsequent fuller immunity.

Die Minister van Gesondheid, sy Ed J. F. Naudé, het die volgende verklaring op 4 November 1955 uitgereik:

Twee hoeveelhede poliomiëlitis-entstof wat deur die laboratoriums van die Suid-Afrikaanse Poliomiëlitis-Navorsingstigting berei is, was vanjaar vir uitreiking beskikbaar. Albei hoeveelhede het die veiligheidstoetse wat ingevolge die jongste regulasies van die Verenigde State se Volksgesondheidsdiens vereis word met welslae deurstaan.

Weens die ongelukkige voorvalle met poliomiëlitis-entstof wat in Amerika gerapporteer is, het die Komitee van Deskundiges wat deur die Minister byeengeroep is om hom in verband met die vrystelling van die Suid-Afrikaanse entstof raad te gee, aanbeveel dat die volledige veiligheidstoetse herhaal moet word op elke hoeveelheid voordat dit uitgereik word, ten einde dubbeld seker te maak aangaande die veiligheid daarvan.

Die eerste hoeveelheid entstof wat toe nog nie in bottels verpak was nie, het die veiligheidstoetse weer met welslae deurstaan en is vir gebruik in September en Oktober uitgereik. Geen ongunstige reaksie is ten opsigte van die talryke kinders wat met die entstof ingespuit is, aangemeld nie.

Die tweede hoeveelheid entstof, wat eers 'n maand na die uitreiking van die eerste hoeveelheid benodig was, is in bottels bewaar nadat 'n chemiese bederfwerende middel bygevoeg is. Dit is gevind dat daardie chemiese bederfwerende middel die uitvoer van die veiligheidstoetse belemmer en as gevolg van daardie tegniese moeilikheid was dit nie moontlik om die herhaling-veiligheidstoetse tot nog toe te voltooi nie. Hierdie moeilikheid kan te bowe gekom word, maar dit sal nog 'n maand duur voordat die resultate van die verdere toetse beskikbaar sal wees. Daar dit dan al diep in die somermaande sal wees wanneer poliomiëlitis die meeste voorkom, sal die moontlikheid van toevallige gevalle van poliomiëlitis wat op inenting volg, groter wees as gedurende ander tye. Die Poliomiëlitis-Navorsingstigting het die Departement Gesondheid dienooreenkomstig in kennis gestel en die Minister het besluit dat dit onder die omstandighede nie raadsaam sal wees om daardie hoeveelheid entstof tans te laat uitreik nie.

Verdere uitreikings van die entstof sal nie voor die volgende winter geskied nie wanneer die moontlikheid van die voorkoms van toevallige gevalle die geringste sal wees.

Intussen kan die publiek daarvan verseker wees dat, soos deur onlangse ondervinding in Amerika geblyk het, die enkele inspuitings wat tot dusver toegedien is, 'n belangrike mate van beskerming behoort te bied en ook dat die vertraging van etlike maande om die tweede inspuiting te gee, nie die ontwikkeling van 'n daaropvolgende voller onvatbaarheid sal beïnvloed nie.

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EDITORIAL

SAFETY FIRST IN ANAESTHETICS

Underlining the establishment by the Council for Scientific and Industrial Research of a bursary the holder of which will be expected to investigate the cause of anaesthetic deaths in South Africa[1] is a report on anaesthetic deaths presented in the United States of America by Beecher and Todd.[2]

These workers submit an analysis of data covering more than half a million anaesthetics and collected over a 5-year period from 10 university hospitals scattered throughout the US. Each of the participating hospitals has an established anaesthetic department engaged in undergraduate and postgraduate instruction, and at each hospital all deaths following an operation or an anaesthetic have been studied by an anaesthetist and a surgeon. Those deaths which were classified as due to the anaesthesia, or to which the anaesthetic contributed, provide the material for this report.

Against the background of a surgical mortality of 1·3% the over-all anaesthetic mortality was 1 in 1,566 anaesthetics. If the method of collecting and classifying the data favoured the over-all picture, it failed to gloss over the startling results of analysis of the use of relaxants, including curare, Flaxedil and Scoline.

Relaxants were used for one case in every 14 for both major and minor surgery. The mortality rate for anaesthesia for minor surgery where relaxants were not used was 1 in 5,071 anaesthetics, but this rose to 1 in 2,314 anaesthetics when relaxants were added to the anaesthetic technique. Similarly in major surgery where relaxants were not used the mortality rate was 1 in 1,270 anaesthetics and this climbed to the alarming ratio of one death in every 192 anaesthetics when relaxants were used for major surgery. There was no statistically significant difference between the mortality rates for good-risk and poor-risk subjects, or for institutions which used relaxants 5 times more frequently than those which were more conservative in their use of these drugs.

It must be emphasized that all the figures quoted in the study are for a special statistical population in which only 10% of the anaesthetics were administered by specialist anaesthetists, although all were supervised

VAN DIE REDAKSIE

VEILIGHEID EERSTE BY NARKOSE

Die verslag wat Beecher en Todd[1] in die Verenigde State van Amerika voorgelê het oor narkosesterftes, onderstreep die oprigting deur die Suid-Afrikaanse Wetenskaplike en Navorsingsraad van 'n Beurs waarvan die houer verwag word om ondersoek in te stel na die oorsaak van narkosesterftes in Suid-Afrika.[2]

Hierdie navorsers ontleed gegewens wat meer as 'n halfmiljoen gevalle van narkosetoediening dek, en wat oor 5 jaar ingesamel is uit 10 universiteitshospitale versprei dwarsdeur die V.S.A. Elkeen van die deelnemende hospitale het gevestigde departemente van narkose, waar studente en nagraadse studente opgelei word. By elkeen van die hospitale is alle sterfgevalle na operasie of narkose, deur 'n narkotiseur en 'n chirurg bestudeer. Sterfgevalle wat geklassifiseer is as die gevolge van narkose, of waartoe verdowing bygedra het, vorm die grondslag van die verslag.

Teen die agtergrond van 1·3 persent chirurgiese sterfgevalle, beloop die totale narkosesterftesyfer 1 op 1,566. Indien die metode van insameling en klassifiseer van gegewens die narkosesterftesyfer meer gunstig laat voorkom, het dit tog nie daarin geslaag om die ontstellende ontledingsresultate van die gebruik van ontspanningsmiddels soos kurare, Flaxedil en Scoline te verbloem nie.

Ontspanningsmiddels was by 1 uit elke 14 gevalle vir beide groot en klein operasies gebruik. By klein operasies waar ontspanningsmiddels nie gebruik was nie, het die narkosesterftes 1 uit 5,071 beloop, maar hierdie syfer het gestyg tot 1 dood uit 2,314 verdowings waarby ontspanningsmiddels wel gebruik is in die narkosetegniek. Insgelyks was die sterftesyfer 1 uit 1,270 narkotiserings by groot operasies waar ontspanningsmiddels *nie* gebruik was nie—die syfer het skrikwekkend gestyg tot 1 dood uit 192 verdowings waarby ontspanningsmiddels by hierdie groot operasies gebruik was. Statisties was daar geen betekenisvolle verskil tussen die sterftesyfers van pasiënte wat goeie, of swak, risiko's was nie, of by hospitale wat 5 keer meer dikwels ontspanningsmiddels gebruik het as dié wat meer konserwatief in hul gebruik van hierdie middels was nie.

Dit moet benadruk word dat alle syfers aangehaal in die oorsig betrekking het op 'n spesiale statistiese bevolking, waarin slegs 10 persent van die verdowings uitgevoer is deur spesialis-narkotiseurs, hoewel alle gevalle onder die toesig van 'n spesialis was. In elk geval, die spesialiste het 'n hoër sterftesyfer as die anestetiese registrateurs behaal (1 op 800 teenoor 1 op 1,200), waarskynlik omdat eersgenoemdes 'n relatief

by specialists. In any event, the specialists showed a greater anaesthetic mortality rate (1 in 800) than anaesthetic registrars (1 in 1,200), probably because the former anaesthetized a relatively greater number of poor-risk patients. Inhalational anaesthesia (the bulk of it with nitrous oxide, oxygen and ether) was used in 44%, intravenous anaesthesia in 22% and local anaesthesia in 30% of the 599,548 anaesthetics which were studied.

The authors do not attempt to explain why 'modern' anaesthetic techniques are so dangerous, but they emphasize that the relaxant drugs may cause circulatory collapse as well as respiratory failure. They point out also that they themselves merely collated information supplied to them at 4-monthly intervals. They returned the collated material, in the form of an annual analysis, to its respective sources, and this service led to incredulity on the part of those originally supplying the data. The authors stress that very commonly 'clinical impressions' are not borne out by observed facts.

In 1851 C. R. Gilman[3] wrote, 'I have laboured to impress on the minds of those (using anaesthetics) as yet unfamiliar with their use, the lessons of caution and watchfulness without which I know these agents are, and must be, *ministers of death*'. Caution and watchfulness! Beecher and Todd's report brings into relief the fact that these are as necessary in the use of relaxants in anaesthesia as they were a century ago when the use of chloroform, ether and nitrous oxide was new.

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groter aantal swak risiko-pasiënte behandel het. Inasemverdowing (meestal met distikstofoksied, suurstof en eter) was by 44 persent gevalle gebruik; aarverdowing in 22 persent en plaaslike verdowing in 30 persent uit die 599,548 gevalle wat die werkers bestudeer het.

Die skrywers probeer nie om te verduidelik waarom die ‚moderne' verdowingsmetodes so gevaarlik is nie, maar hulle lê daarop nadruk dat die ontspanningsmiddels verantwoordelik kan wees vir instorting van die bloedsomloop en van die asemhalingstelsel. Hulle meld dat hulle eintlik die gegewens wat hulle elke 4 maande ontvang het maar net verwerk het. Hulle het die saamgestelde gegewens, verwerk tot jaarlikse ontledingsverslae, aan die onderskeidelike bronne teruggestuur. Hierdie diens het groot ongeloof as gevolg gehad onder die persone wat oorspronklik die gegewens verskaf het. Die skrywers beklemtoon die feit dat ‚kliniese indrukke' baie dikwels nie deur waargenome feite gestaaf word nie.

In 1851 het C. R. Gilman[3] geskryf: ‚I have laboured to impress on the minds of those (using anaesthetics) as yet unfamiliar with their use, the lessons of caution and watchfulness without which I know these agents are, and must be, *ministers of death*'. Versigtigheid en waaksaamheid! Die verslag van Beecher en Todd beklemtoon die feit dat hierdie twee voorsorgmaatreëls vandag by die gebruik van ontspanningsmiddels nog net so onontbeerlik is as 'n honderd jaar gelede toe die gebruik van chloroform, eter en distikstofoksied nog iets nuuts was.

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DIAGNOSTIC TESTS IN RHEUMATIC DISEASES

The term 'rheumatic diseases' includes a large number of conditions. Several new tests have been developed in recent years to aid in their diagnosis.

In rheumatic fever new tests have been devised on the basis of the preceding haemolytic streptococcal infection. In diagnosis many investigators estimate the antibody titre in the serum, which is elevated in most patients with rheumatic fever;[1] the streptococcal toxin named streptolysin-O is involved in the test, so that this test is referred to as the antistreptolysin-O determination. A rising titre in a series of readings is more significant than a raised figure in a single estimation. There are similar tests which determine anti-hyaluronidase, antistreptokinase, and antidornase.

The determination of the erythrocyte-sedimentation rate is useful in following the course of infectious diseases, and is much used in rheumatic fever. It is usually insisted that a patient who has had rheumatic fever is not to be allowed out of bed until the ESR is normal or near normal. The ESR may remain high for weeks after other evidence of the disease has disappeared. In these patients the C-reactive protein test is helpful. Like the ESR the C-reactive protein test is non-specific and its greatest value is in following the course of the disease,[2] although like the ESR it is useful in differentiating mechanical disabilities like degenerative joint-disease and traumatic arthritis from arthritides associated with active inflammation. The test is based on the presence in the blood of a protein not normally present and which, quite coincidentally, is able to precipitate pneumococcal C-polysaccharide. It is to be noted that, unfortunately, the two tests are not reliable when corticotrophin or steroids are given, because of their anti-inflammatory action which masks underlying active disease.

The C-reactive protein test is easier to carry out than an ESR determination but otherwise has no special advantage. Specific tests of activity such as agglutination tests are being examined, but are difficult to carry out and not widely used.

In rheumatoid arthritis the sedimentation rate is used to determine the presence of active disease, which may continue when clinical observation shows remission of joint inflammation.

Disseminated or systemic lupus erythematosus may now be diagnosed by the aid of the LE test, with the

result that the diagnosis of this condition is being made more frequently than formerly. A serum protein which resides in the gamma-globulin fraction is believed to liberate intracellular desoxyribonuclease, which depolymerizes nucleoproteins of the nucleus;[3] hence the appearance of a homogeneous basophilic inclusion in the cytoplasm of polymorphonuclear neutrophilic granulocytes (the LE cell). The ESR is high in this disease.

In gout the uric-acid level in the serum is elevated, and reflects the increased pool of urate present in gouty subjects. The total amount of urate in the body is increased even in patients who have no demonstrable tophi. The erythrocyte-sedimentation rate is increased in gout and C-reactive protein is present.[1]

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CONGENITAL DUODENAL OCCLUSION WITH A REPORT ON FIVE CASES SUCCESSFULLY TREATED

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Congenital duodenal occlusion is a condition in which there is an interruption in the continuity of the lumen of the duodenum from a point just above, opposite or below the entrance of the common bile duct—the so-called Draper's dead area—to the duodeno-jejunal flexure. The occlusion may be complete or incomplete, and the casue intrinsic or extrinsic. Duodenal atresia is met with in the second part, and is due to some intrinsic cause such as a complete diaphragm, or a blind termination either totally disconnected from the rest of the alimentary tract or connected to it by a fibrous cord. Symptons manifest themselves at or within a few hours of birth. Duodenal stenosis, representing a partial obstruction of varying degree, usually occurs in the third part or at the duodeno-jejunal flexure. Extrinsic cause is the rule, such as compression by abnormal developmental bands or by faulty fixation of the midgut loop with or without volvulus. Occasionally an intrinsic fault may be found, e.g., an incomplete diaphragm, but partial duodenal occlusion is the common variety. Symptoms manifest themselves for the first time days, weeks or months after birth. Duodenal occlusion may occur alone, or in association with other congenital anomalies. A particular association with mongolism occurs.

We report here 1 case of duodenal atresia and 4 cases of duodenal occlusion successfully operated upon.

History. Calder in 1752 described a case for the first time. The first attempt at surgery was undertaken by Voison in 1804, a truly herculean effort in pre-listerian and pre-anaesthetic days. He performed an unsuccessful gastro-enterostomy. Not until 107 years later was the first successful operation performed, by Fockens[1] in 1911, to be followed by Ernst[2] in 1916 with the second successful gastro-enterostomy, in an 11-day-old child. Throughout all the preceding years most of our knowledge was obtained from post-mortem specimens. Even most of Louise Corde's cases, numbering 57, were reports on autopsies. From 1916 reports on successful cases suddenly appeared from numerous sources, though few observers were able to report more than one or two cases individually. Donovan[3] reported 12 recoveries out of 18 cases operated on. Then came Ladd,[4, 10] a pioneer in paediatric surgery, who was able in 1939 to collect 39 cases of his own, with 26 recoveries. He was followed by Gross,[5] who in 1940-1952 reported on 55 cases with 36 recoveries. Forshall,[6] in one of the most comprehensive and informative articles on this subject, reviewed all the published cases up till 1947. She contributed 4 of her own cases. The credit for the first successful case operated upon in South Africa belongs to Jacobsz,[7] who, in February 1949, performed a posterior gastro-enterostomy on a 5-day-old baby. Swain[8] recently reported 5 recoveries out of 7 cases over a period of 6 years.

Incidence. Many cases die without surgery or autopsy, and statistics can therefore only be approximate. In 1877 Therimim reported an incidence of 1 in every 20,000 births but, with a greater awareness of this condition, the reported incidence has been brought up to 1 in 6,000 births. Somerville[9] reported an incidence of 1 in 2,000 post-mortems. At the Children's Hospital, Johannesburg, during the 6 years ending 1953 inclusive, out of 24,000 admissions 10 cases were diagnosed and confirmed by operation or autopsy. Four of the cases recorded in this article are included in the total of 10.

Embryology. At about the 10th or 11th week of development, two events take place relevant to the condition under discussion, viz. (1) the conversion of the alimentary tube from a solid to a hollow organ, and (2) its return from the umbilical cord to the coelom and its subsequent rotation and fixation. Errors in the process of vacuolation may lead to the formation of a

diaphragm in the duodenum, or its complete separation into two portions with or without a connecting fibrous cord. Errors in the return of the tube from without to within the coelom will lead to non-rotation or mal-rotation of the gut and, in the final disposition, to the development of abnormal bands, often in association with volvulus of part or all of the midgut loop. The twist is usually in a clockwise direction, and the blood supply is only rarely involved.

CLINICAL PICTURE

In the *atretic* type, where the obstruction is of the intrinsic variety, vomiting is the presenting symptom and occurs within 24 hours of birth, soon becoming projectile. Since in the great majority of cases of duodenal atresia the obliteration is just distal to the bile papilla, the vomitus contains bile in varying amounts. The first stool may consist of meconium, but this is soon replaced by mucus only. In the occasional case where the obstruction is proximal to the termination of the common bile-duct the vomitus will be free of bile and the stool contain bile and mucus. The infant goes downhill rapidly from dehydration, ketosis, aspiration pneumonia and starvation. Death occurs within 7-9 days. On examination in the early stages physical signs may not be obvious. There may be some fullness in the upper abdomen, with a scaphoid lower abdomen. Peristaltic waves in the upper abdomen from left to right may be seen, and occasionally in the reverse direction. A tumour is not felt as in congenital hypertrophic pyloric stenosis. Rectal examination is not helpful. A straight X-ray of the abdomen in the erect and oblique positions is of the greatest help in diagnosis. This will show dilatation of the stomach and a variable portion of duodenum without demarcation, and with a fluid level in one or both. Gas is absent in the rest of the intestine. Administration of barium is usually unnecessary; it harbours the great risk of fatal barium pneumonitis from regurgitation and inhalation.

In the *stenotic* type the features are of lesser gravity and in direct proportion to the degree of stenosis. Thus the vomiting may be delayed in onset and intermittent in nature and may represent only part of the food intake, so that nutrition may be maintained. It is characteristically bilious. Meconium will be passed—and, later, normal, if smaller, stools. At times the infant appears well, with complete freedom from symptoms, but in the long run a downward course is initiated, progressing to the usual manifestations of malnutrition. Physical signs in the early stages may be absent. Sometimes not only the upper abdomen, but the whole of the abdomen, may become distended without any localizing signs. Later, loss of weight and wasting become evident. Here again the best guide to diagnosis is a straight X-ray. Since the lesion is in the third part of the duodenum, this latter viscus will appear widely dilated, with a fluid level in addition to that present in the stomach, giving the picture of 'double bubble'. The effect of stagnation may be such that the duodenum appears more widely distended than the stomach. In addition there will be seen gas scattered in the small and large bowel and evidence of non-rotation or mal-rotation may be evident. The position of the caecum can be demonstrated by an enema. In these cases the administration of a small quantity of radio-opaque material by mouth is permissable, because the obstruction is partial, the patient older, and the consequent risk of inhalation minimal.

TREATMENT

Pre-operative. This will vary as between the two types. In the atretic type, as soon as the condition is diagnosed every effort must be made to combat the dehydration and to decompress the stomach. A period of anything up to 24 hours spent in correcting these is time well spent and well rewarded. Elaborate tests need not be undertaken, but fluid, blood and plasma in proportion to the infant's weight must be given intravenously in the usual manner. Intermittent gastric suction through a small soft rubber catheter suffices to empty the stomach.

The position is somewhat different in the incomplete type, whether due to extrinsic or intrinsic cause. Relief from obstruction may be required in the first week or two of life, but in other circumstances, e.g., in premature babies where the condition is such that the patient is able to put on weight despite intermittent vomiting, it may be found advisable to wait a few weeks until the weight and maturity of the infant render the operation less hazardous. This approach was adopted in our case 3.

Operation

Anaesthetic. Atropine premedication is given in the usual way, followed by ethyl chloride and ether sequence. An endotracheal tube, preferably an Ayres flexometallic type, is introduced. Thereafter a small rubber catheter is passed into the stomach. Anaesthesia is maintained by insufflation with nitrous oxide, oxygen and ether mixture, applied with positive pressure where necessary.

The tube in the stomach serves a double purpose. It keeps the stomach empty of fluid contents, thus preventing aspiration, and at the request of the surgeon enables the anaesthetist to inflate the stomach and duodenum down to the point of obstruction, thus facilitating the surgical procedure.

Surgical Procedure

A very generous incision, right paramedian, either lateral rectus displacing or rectus splitting, is a *sine qua non.* It must be so large that all non-fixed viscera literally fall out on to the abdominal wall, and inspection can be effected with the minimum of handling. The disposition of the intestines, the small in relation to the large, the colour and calibre, will give the clue to evidence of mal-rotation, and the presence of a volvulus. The spreading of the intestine itself on the abdominal wall will make the detection of developmental bands easier. Depending on the nature of the pathological condition, any of the following procedures may have to be undertaken, separately or in combination:

1. Gastro-jejunostomy (anterior or posterior). This procedure is recommended for obstructions situated in the middle of the second part of the duodenum. If a posterior gastro-enterostomy is decided upon, no time need be wasted in suturing the margins of the rent in the transverse mesocolon to stomach; this may seem

heretical, but it is good practical advice. In all other respects the operation proceeds as in the adult. Clamps are dispensed with. The anastomosis is effected by means of an outer row of interrupted 0000 sutures, of non-absorbable material and an all-coats continuous double-nought 00 20-day chromic catgut on an atraumatic needle. Bleeding points are caught and tied. This procedure was employed in our one case of atresia (case 1). Nothing more elaborate is needed than this simple procedure. It meets the case, and is not time-consuming.

2. A retro-colic duodeno-jejunostomy for obstructions in third part of duodenum and at the duodeno-jejunal flexure. All four of our cases of incomplete obstruction were treated in this manner.

3. Excision or transection of abnormal peritoneal bands. To do this as the sole procedure requires experience, and courage, but it has its application. The commonest finding is obstruction of the descending portion of the duodenum by a broad tight peritoneal band tethering the undescended caecum in the right hypochondrium to the lateral abdominal wall. Incision or excision of this band, which does not contain any vessels to the intestine or colon, releases the obstruction. The caecum falls over to the left and downwards, displaying a duodenum which extends vertically downwards, devoid of flexures, and passing insensibly into jejunum. Ladd was the first to draw attention to this condition, and the first to suggest this method of treatment. We tried the method in one case (case 5). About 14 days later it was necessary to re-open the abdomen and supplement it with a duodeno-jejunostomy.

4. Untwisting a volvulus. It may be necessary to supplement this with a duodeno-jejunostomy, or Ladd's procedure.

Post-operative care brings its special problems of control of water, electrolytes, blood and protein. One of the most troublesome complications is persistence of of bilious vomiting, due to oedema of the stoma. This, in turn, is usually due to the excessive administration of salt in the intravenous drip. For the first 48 hours, therefore, the salt content should be kept low. Water and blood requirements are controlled in the usual way, and it is wise to employ an antibiotic. Aspiration of stomach contents, either continuous or intermittent, is practised for the first 24-48 hours after the operation. For our part, we prefer intermittent suction with a catheter and syringe, 4-hourly. The entire procedure takes a couple of minutes and the catheter is withdrawn. The quantity withdrawn is measured, and when it is less than 15 c.c. suction may be discontinued.

CASE REPORTS

Case 1. *Duodenal Atresia*

G.B., white male child, normal delivery. Birth weight 6 lb. 4 oz. Admitted when 4 days old on 11 September 1948 weighing 5 lb. 8 oz. The baby had vomited almost incessantly from birth and the vomitus contained bile. No meconium has been passed. Considerable dehydration was present. Examination showed a distended upper abdomen. No peristalsis was noted. Straight X-ray showed a dilated stomach and duodenum with a single fluid-level. No gas was seen in the rest of the alimentary tract. A diagnosis of duodenal atresia was made, and 24 hours were spent in correcting dehydration with intravenous glucose in water, to which was added vitamins B and C and salt.

Operation at age of 5 days. 60 c.c. of whole blood were given during the operation. The stomach and the proximal half of duodenum were found to be greatly dilated. Below this point the bowel was collapsed and resembled a $\frac{1}{4}$ inch ribbon. The stomach when collapsed after suction through a gastric tube was the size and shape of a hen's egg. Posterior gastro-jejunostomy through a $1\frac{1}{2}$ inch stoma midway between cardio-oesophageal orifice and pylorus was carried out. The abdominal incision was closed in layers. It is interesting to note how the stoma had drifted to the right when seen $6\frac{1}{2}$ years later (Fig. 1).

Fig. 1. Case 1. A supine film taken 6 years post-operatively, demonstrating a posterior gastro-enterostomy; the site of the gastro-enterostomy has been indicated by markers. Note the absence of filling of the duodenal cap and first part of the duodenum.

Post-operatively. A catheter was passed every 4 hours and the stomach contents aspirated. Intravenous infusion of glucose water and later saline was maintained for 4 days, after which stomach suction was discontinued and oral feeding instituted. Recovery was uninterrupted, and the infant was discharged 15 days after the operation weighing 6 lb. 6 oz. At the age of 6 years the child is well. He is well grown and well nourished, and in all respects a normal child.

Case 2. *Duodenal Stenosis*

A.L., an Indian male, birth weight 6 lb. 4 oz., was seen on 1 July 1949 at the age of 6 weeks, weighing 6 lb. The infant had started vomiting when 1 week old, when a diagnosis of pyloric stenosis was made by a practitioner and Eumydrine given without response. The vomits were copious and bile-stained, and occurred for the main part just after the feeds. The infant took his feeds eagerly. The bowels were constipated. At the age of 6 weeks, the infant was thin and shrunken, with his skin hanging in folds, but he was neither toxic nor ill. On feeding, peristalsis was visible across the abdomen from left to right, then from right to left followed

by bilious vomiting. A clinical diagnosis of duodenal obstruction was made, and the child referred to hospital for operation.

X-ray Report. 'Control radiograms in erect position show distension of stomach with horizontal fluid-level due apparently to retained stomach contents. A little gas is seen in the position of the first part of duodenum, but it is not distended. There is only a small amount of gas in the rest of the bowel. After 20 c.c. of barium through a catheter passed into stomach, obstruction of the duodenum was demonstrated in its horizontal part. Films taken 20 minutes later show that only small traces of barium have passed beyond point of obstruction'. The diagnosis of duodenal stenosis was thus confirmed and the child prepared for operation.

Operation on 2 July 1949. On laparotomy the duodenum was found to be obstructed at the duodeno-jejunal flexure. This was better demonstrated when the stomach was inflated by the anaesthetist. The air was arrested at the obstruction, distending the duodenum. At this point the duodeno-jejunal area was twisted upon itself by peritoneal bands. The bands were severed, the twist of 360° undone, but very little air would pass beyond on attempts being made to blow down the catheter, even though the stomach and duodenum became greatly distended. As soon as the intestine was released, the twist recurred. Without wasting any more time, a retro-colic duodeno-jejunostomy just proximal to the obstruction was performed. The distended duodenum made the surgical procedure a relatively simple matter. The abdomen was closed in layers. The patient was returned to the ward in good condition.

Fig. 2. Case 2. Supine film taken after the administration of a barium meal 5 years post-operatively showing a duodeno-jejunostomy. The site of anastomosis has been indicated by an arrow. Note the absence of any dilatation of the duodenum proximal to the anastomosis.

Post-operative treatment was along the usual lines. Convalescence was uneventful. The child was discharged on the 24th day weighing 7 lb. He is well 5 years later.

Case 3. Duodenal Stenosis

L.G.L., a first child, female, 2 weeks premature. Birth weight 5 lb. 8 oz. Vomiting commenced on 1st day of life. Meconium passed normally. Seen for first time on 8th day, temperature 104°F, dehydrated and having been convulsed the same day; weight 5 lb. Vomiting had been intermittent, but usually bile-stained. Constipated with occasional loose stool. Had fed well throughout. A provisional diagnosis of duodenal stenosis was made; pyrexia was thought to be due to dehydration, and the convulsion to electrolyte imbalance consequent on chloride loss. The general condition of the child was not disturbing once these losses had been made good with intravenous saline. Admitted to hospital 2 days later on 23 August 1950 in good general condition. Good colour, no anaemia, no dehydration or oedema. The abdomen was not distended but there was visible peristalsis in the upper half to the left of the midline only. The large bowel was heavily laden with faeces and easily palpable. Straight X-ray suggested duodenal obstruction. Vomiting was minimal at this stage and only occasionally bilious. Normal feeding was persevered with. At the age of 19 days (weight 5 lb. 8 oz.) a barium meal was administered.

X-ray Barium Meal. 'The duodenal obstruction previously reported is still present. The proximal duodenal loop is dilated, and shows considerable writhing movements, making localization of obstruction difficult, but it is believed to be in third portion. The stricture is best demonstrated on the 5-hour film, which also shows considerable residue in the stomach, and particularly in the proximal loop. At 24 hours there is barium in the duodenal loop. The degree of stenosis suggests that this is an intrinsic stenosis, rather than one due to pressure of the mesenteric artery.'

As the child was gaining weight slowly, there appeared no need for immediate operation, and it was at the age of 9 weeks, with the weight at 7 lb., that the infant was considered suitable for surgery.

Operation. Retrocolic duodeno-jejunostomy just proximal to obstruction at duodeno-jejunal flexure. Site of obstruction well demonstrated by inflating stomach and duodenum with air. Cause of obstruction obviously intrinsic. The patient left the

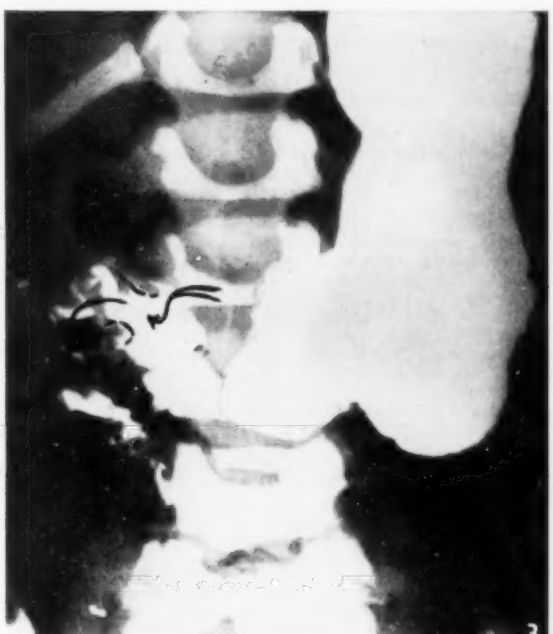

Fig. 3. Case 3. Supine film 4 years post-operatively demonstrating a duodeno-jejunostomy. The site of enterostomy and the course of the barium is indicated by arrows.

table in good condition and made an uneventful recovery, taking her feeds well, with bowels acting normally. She was discharged 14 days after the operation. She has remained trouble-free and at the age of 4 years weighs 33 lb.

Case 4. *Extrinsic Obstruction*

P.A.K., a case of incomplete development and incomplete fixation of mesentery; chronic volvulus.

First admission on 9 October 1947 at the age of 7 months. Birth weight 6 lb. Well until 2½ months old, when he developed projectile vomiting about 2-3 hours after feeds. Some vomits were bile-stained. A diagnosis of pyloric stenosis was made by a practitioner, who prescribed Eumydrine with almost complete cessation of vomiting. The infant picked up well until 6 months

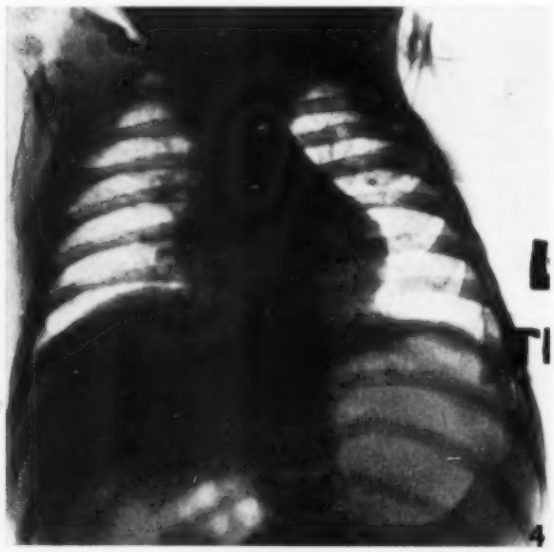

Fig. 4. Case 4. Teleradiogram taken 1 year before operation. A gross dilatation of the gas-filled stomach and duodenum is visible but the importance of this finding was not appreciated at the time.

old when he went off food and started vomiting again. The vomitus was malodorous and bile-stained; stools loose, frequent, slimy, pale and foul-smelling. He was subject to frequent colds and a 'rattly' chest, at which times a cough was present.

On examination. Weight 14 lb., had lost 1 lb. in a vomiting episode 1 week before admission. Nothing abnormal found in heart and lungs. The abdomen was doughy and not distended. Rest of examination negative. Blood count normal. ESR 2. Blood chemistry: Calcium 10·6 mg.%, phosphorus 4·3 mg.%, Protein 5·2 g.% (albumen 4·6, globulin 0·6). Nothing abnormal in urine. Stool: 78% water, 31% fat—normally split—no starch detected.

X-ray. See Fig. 4. On this plate, reviewed 2 years later, the 'double-bubble' of duodenal obstruction is clearly visible, but no comment was offered by the radiologist. The wrists showed a normal ossification index for the child's age.

On admission he was given skimmed milk and Casec. When starch was introduced he developed severe diarrhoea and vomiting, and required intravenous therapy to restore hydration. He was thought to be a possible case of early coelic disease. Discharged after a week weighing 14 lb. 8 oz., representing a gain of 8 oz. in hospital. Duodenal obstruction was not suspected.

Second admission on 2 March 1948 when 1 year old. Had kept well for 4 months and gained weight to 16 lb., when he had a sudden vomiting attack. A week later vomiting recurred, and the same evening the child passed a fair amount of blood in the stool, at which stage he was admitted to hospital. The episode was regarded as one of gastro-enteritis, although the vomiting and loose stools settled quickly.

Third admission on 8 April 1948 when 13 months old. Admitted with acute pharyngitis, which settled on sulphadiazine. Discharged in 4 days. No diarrhoea or vomiting. On reviewing the case, the possibility of duodenal obstruction was suspected for the first time and arrangements were made for a barium meal and enema.

20 *April* 1948. X-ray and barium meal—passed as normal. No comment on duodenum. No evidence of any hold-up or ileus. Barium enema—high caecum.

Fourth admission on 30 June 1948, when 15 months old. The child was sent for and re-admitted for further radiological study. Weight 16 lb. 8 oz. The X-rays taken 2 months before had suggested to us the possibility of duodenal compression because of a high, undescended caecum.

Barium meal report: 'Oesophagus and stomach normal. Follow-through indicated a great deal of intestinal hurry. Lower ileum reached in 1 hour; sigmoid in 2½ hours. Considerable pooling of the meal in the small bowel—consistent with neuromuscular dysfunction often associated in infancy with nutritional disorder'. Once again it was not possible to show duodenal obstruction.

2 *October* 1948. Barium enema performed for mal-rotation of caecum. It was decided that the caecum was not mal-rotated. Two further examinations followed in quick succession, the radiologists having difficulty in coming to a diagnosis. A barium enema revealed a displacement of most of the sigmoid colon to the right side, the caecum appearing to be upturned.

4 *November* 1948, aged 20 months. 'A barium meal revealed a fairly dilated second portion of the duodenum in which definite reverse peristalsis was noted (Fig. 5). Once past the second part

Fig. 5. Case 4. Erect film taken pre-operatively after the administration of barium, demonstrating two fluid levels; one in a dilated stomach and one in the duodenum.

of the duodenum there was hypermotility, the opaque medium reaching the rectum in 4 hours. Caecum again showed to be undescended and overlying the third part of the duodenum'. At this stage the diagnosis of duodenal obstruction had been agreed on, but vomiting attacks had ceased and it was resolved to leave surgery in abeyance.

Throughout 1949 the child kept well, until October.

Fifth admission on 13 January 1950, at the age of 2 years and 10 months. Vomiting attacks had recurred every 3 weeks or so in the past 3 months and were preceded by abdominal distension; sometimes abdominal pain in attack. Weight 28 lb. (normal —33 lb.). Height 35½ inches (normal—37 inches). Prominent abdomen and small buttocks.

A barium meal showed increased distension of duodenum. Operation was suggested at this stage, but was refused. Eight months later, because of persistence of attacks of vomiting, operation was agreed upon.

Final admission. Operation was performed on 27 September 1950 at the age of 3½ years.

Chronic volvulus of entire small intestine, through 3 complete turns in a clockwise direction. The intestines were darker in colour than normal and only slightly dilated. This volvulus was due to a lack of development and consequent lack of fixation of the mesentery of the small intestine, which was represented by a small pedicle only at its extreme upper end at the duodeno-jejunal flexure. The mesentery had the appearance of a wedge, broad at its posterior attachment, measuring 1 inch laterally, and tapering towards the intestine. It was of the consistency of india-rubber and the main blood supply coursing through this wedge to the intestine could not be palpated. It appeared to be obstructing the termination of the duodenum, which was greatly dilated. Coursing over the surface of the third part were numerous large engorged veins. Some of these veins had to be transected in order to obtain an area large enough to perform a retrocolic duodeno-jejunostomy. The volvulus was easily untwisted, and a duodeno-jejunostomy performed. The patient made an uneventful convalescence and was discharged 14 days after the operation.

One month later he weighed 33½ lb. One year later, when 4 years and 9 months old, he had put on 8 lb., weighing 41½ lb.; good appetite, looking well, normal abdomen, one stool daily and no vomiting. Aged 7 years, he is a strapping boy with normal digestion.

Case 5

P.B., seen at his birth in August 1949, when a diagnosis of mongolism was made. Birth weight 8 lb. 3 oz. Heart sounds closed. At the age of 2 months, when the weight was 10 lb. 2 oz., was reported to be possetting a great deal. The infant continued to thrive, and when seen at the age of 6 months for an intercurrent diarrhoea, the weight was 17 lb. A systolic murmur of moderate intensity was present. At the age of 13 months the weight was 20 lb. 8 oz., and the infant was taking solids but was still inclined to vomit. Up to this stage the degree of vomiting was not such as to be regarded by the mother as a major complaint. At 18 months the weight was 22 lb. 2 oz., and the vomiting had become more pronounced. For the first time the possibility of an intestinal obstruction was mooted but, as the child was resentful of examination, the barium examination was postponed at the parent's request. A diastolic element was now noted in addition to the systolic murmur and a diagnosis of patent ductus was made. At the age of 21 months the vomiting had become explosive, and the abdomen was slightly prominent; weight 24 lb. 9 oz. A typical Gibson murmur was present in the heart. At this stage the barium examination was permitted by the parents.

X-ray on 12 May 1951. The stomach was grossly distended and in the erect position showed a large fluid-level. The duodenum was distended as far as the junction of the second and third portion. Barium passed this obstruction and at 5 hours complete emptying had occurred. There were two separate fluid levels, the larger one lying on the left in the stomach and the smaller one on the right in the second portion of duodenum. The radiologist's differential diagnosis was: (*a*) 'An extrinsic pressure, such as aberrant pancreas or band', or (*b*) 'A congenital membrane partially occluding the second portion of duodenum'.

Operation was performed on 23 May 1951. Many abnormal bands were found and freed. One broad band was found kinking the second part of duodenum, so that when the latter was insufflated with air it swelled up greatly, demonstrating the site of obstruction with some clarity. The band was cut across and air passed rapidly from duodenum to jejunum. It was felt that the obstruction had been completely relieved. The abdomen was then closed.

All went well until 14 days later, when the child began to vomit, and belched foul winds. The following day a thin barium emulsion was given and the radiologist reported as follows: 'The duodenum was greatly distended. The duodeno-jejunal flexure could not be seen on left side of spine. Very slow emptying of duodenum. At 1 hour there was still en enormous residue present in stomach and duodenum. The small bowel lay on the left side of the spine but the duodeno-jejunal flexure appeared to be on the right side'.

Re-operation on following day: Stomach evacuated. Healed incision excised. Duodenum widely dilated. With finger in widely opened third part of duodenum, a perforated diaphragm was found. A retrocolic duodeno-jejunostomy was performed. Child returned to ward in good condition. Three days after the operation the bowls acted normally. Except for a slightly infected abdominal wound, and evidence of thrombo-phlebitis, the result of venoclysis, the little fellow made an uneventful recovery.

Three years later he was still free from any abdominal symptom and the digestion was normal.

DISCUSSION

A review of the reported cases suggests that duodenal atresia should offer no real diagnostic difficulty; the urgency of the vomiting compels immediate attention. Partial duodenal occlusion is of quite a different order. The vomiting may come on any time from birth to the age of 1 year or later. It may be bilious, copious, continuous and projectile, but may also be non-bilious, intermittent and 'possetting' in nature. The vomiting attacks may be months apart. The condition is consistent with good health and weight, and on the other hand may result in starvation and wasting. In one of our cases the vomiting occurred in explosive bouts lasting a few days with a goodly interval between, resulting in the pattern seen in cyclical vomiting. The attacks were accompanied by diarrhoea, weight loss and dehydration. Abdominal distension, wasting, small buttocks, interference with growth, and large foul starch-containing stools, made the similarity to coeliac disease uncomfortably close. The need for routine barium-meal examinations in all such cases of persistent vomiting, diarrhoea or failure to gain is now fully appreciated.

In the ordinary way, surgery will be instituted as soon as the diagnosis has been made, always allowing for pre-operative hydration. With partial obstruction in very small neonates or prematures, where the condition is insufficient to interfere with gain in weight, it might be advisable (as in case 3) to wait a week or two until the infant is of a satisfactory weight to withstand a major operation.

As to the operation itself, gastro-jejunostomy or duodeno-jejunostomy is both safe and satisfactory. From time to time, it would appear from the literature that it suffices to cut abnormal bands and to free the caecum if incompletely rotated. However, case 5 taught us a sharp lesson. It appeared at the time that by cutting bands the duodenum had been completely freed. However, two weeks later it became necessary to do a short circuit. At operation an incomplete diaphragm was found in the lumen of the duodenum.

The post-operative treatment, a matter of fine balance of water, blood, plasma and electrolytes, can be most trying. In these small infants the margin between hyper- and hypo-electrolytaemia is small. In this connection it is well to remember that hypernatraemia and hyperkalaemia are man-made diseases, and that in the field of intravenous therapy over-hydration is commoner than under-hydration.

SUMMARY

1. Congenital duodenal occlusion is reviewed as regards its history, incidence, embryology, clinical picture and treatment. The atretic and stenotic types are distinguished.

2. One case of duodenal atresia and 4 cases of duodenal stenosis successfully operated on are reported.

3. Duodenal stenosis may cause difficulty in diagnosis because of long intervals between the vomiting bouts. Minimal vomiting and long-continued diarrhoea may result in a picture resembling the coeliac disorder.

4. Posterior gastro-enterostomy was carried out in 1 case, and duodeno-jejunostomy in 4 cases. An attempt in 1 case to limit the operation to separation of adhesions was unsuccessful.

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THE OCCURRENCE OF WEST NILE VIRUS IN SOUTH AFRICA*

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West Nile virus received its name because it was first isolated from the blood of a Native woman who resided in the West Nile district in Uganda. A blood specimen was taken from this woman because she was found to have fever during a routine examination which was part of a sleeping-sickness survey. Though febrile, she denied having any symptoms of illness, possibly because of a desire to avoid hospitalization. The first sample, which yielded the virus, contained no neutralizing substances for it; but the second one, collected some 3 months later, neutralized strongly.[1]

West Nile virus is pathogenic for mice by intracerebral, intranasal, and intraperitoneal routes of inoculation. When inoculated intracerebrally it induces a fatal encephalitis in rhesus monkeys; when inoculated peripherally into rhesus monkeys, or by intracerebral or peripheral routes into *Cercopithecus* monkeys, it stimulates the production of antibodies without inducing any clinical signs of illness.[1, 2] Rabbits, guinea pigs, and hedgehogs also produce antibodies to the virus but show no signs of illness.

Antigenic relationship has been demonstrated between this virus and the viruses of Japanese B, St. Louis,[3] and Murray Valley[4, 5, 6] encephalitides, which cause severe, and sometimes fatal, illness in man. Southam and Moore,[7] using various strains of West Nile virus to infect man experimentally, have shown that it is capable of causing encephalitis.

The fact that West Nile virus attacks man and has a wide distribution is shown by the presence of neutralizing antibodies to it in human sera collected in the Anglo-Egyptian Sudan, Kenya, Uganda, the Belgian Congo,[8] Egypt,[9] India,[10] and Israel.[11, 12, 13]

Until recently, little was known of the symptoms the virus produces in man following a natural infection. Now, however, adequate descriptions are available[11, 12, 13] of the illness as seen in Israel between July and September of 1950, 1951, and 1952 in epidemics which were conclusively proved to be due to West Nile virus. The main features of the disease, as reported in the papers just cited, are summarized below:

The disease has a sudden onset of fever, which may reach a maximum of about 104°F but never exceeds this level. The temperature remains elevated for about 72 hours before slowly returning to normal. This usually occurs between the 7th and 9th days. In approximately 10% of the cases[13] there is a recurrence of symptoms and a second bout of fever of 3-5 days' duration occurring 1-3 days after the initial fall of temperature to normal. At the onset of the illness, and occasionally accompanying the fall in temperature, there may be bradycardia (Newman and Southam[14] have shown pathological changes in, and have re-isolated the virus from, human heart muscle). In 78% of the adults and 37% of the children there is severe frontal headache, which is often accompanied by pain when the eyes are moved. The majority of adults complain of malaise and muscular pain, and about 20% of the cases also have abdominal pain. Nausea and anorexia are more common in adults, while diarrhoea and vomiting are more common in children. Bernkopf *et al.*[13] noted that although 45% of the children vomited, the figure for adults was only 19%. Goldblum *et al.*[12] noted that general weakness, drowsiness, and flushed face were frequently seen, although these symptoms are not mentioned by the other authors. All agreed, however, that there was likely to be scleral and pharyngeal infection and that there were many complaints of sore throat, mainly from adults.

Bernkopf *et al.*[13] noted that 5·7% of the cases had severe angina, 2 cases being so severe that they were suspected of having diphtheria. During the first 3 days of the disease the majority of children and a few adults developed a maculopapular rash, with bright pink, round, discrete macules, and slightly raised papules 2-4 mm. in diameter. This rash usually appeared first on the trunk and then spread to the face and extremities, persisting for 5-7 days without desquamation.

Many adults, and fewer children, exhibited a generalized enlargement of lymph nodes; no particular area seemed to be singled out for this phenomenon. The

* The investigations upon which this work is based were financed jointly by the Poliomyelitis Research Foundation (South Africa), the South African Institute for Medical Research, and The Rockefeller Foundation, and were conducted under the sponsorship and with the collaboration of the Union Health Department and the Veterinary Division of the Department of Agriculture.

glands—which were firm, discrete, and non-tender—occurred singly, in groups, or in chains, and varied from the size of a pea to that of a cherry. The histological picture of biopsy material from affected lymph nodes was reported by Eylan[11] to be one of non-specific hyperplasia with a hypertrophic reticulum. This condition sometimes persisted for as long as 2 months after the onset of the disease. It is worthy of note that it was the exception rather than the rule to see rash and lymphadenopathy occurring together. Goldblum *et al.*[12] reported occasional enlargement of the spleen, but this was not mentioned by the other authors.

From the histories of visitors and new arrivals in the epidemic areas, it was determined that the incubation period was probably at least 3 days and not more than 6. Rates of attack by West Nile virus were quoted by two authors for groups of 357 and 303 individuals. These rates tallied closely, and when reduced to round figures they read as follows:

Total individuals attacked	40%
Children	
aged 0 to 2 years	100%
aged 0 to 5 years	85%
aged 0 to 15 years	70%
Adults (over the age of 15)	22%

Convalescence in children was fairly rapid. In adults it was much slower, at times lasting several weeks and being characterized by fatigue and lassitude.

Sequelae have not been reported; complications were limited to mild and transient meningeal irritation, as shown by neck stiffness and a positive Kernig's sign in adults, and a positive Brudzinski's sign in children. Bernkopf[13] reported a positive Brudzinski's sign in 10 of 70 children below the age of 5.

Routine laboratory investigations were not of great assistance in these outbreaks. The typical blood-count showed normal red-cells but a mild leucopenia with a relative lymphocytosis and a shift to the left in the polymorphs. The only other abnormal finding was a rise in protein (Pandy) and cells in the CSF taken from those cases which exhibited neurological signs.

Taylor and Hurlbut[15] have isolated the virus from *Culex* mosquitoes in Egypt; and Work, Hurlbut, and Taylor[16] have reported the isolation of the virus from two avian species (hooded crow and rock pigeon) in the same area. During the outbreaks in Israel the virus was isolated from *Culex molestus* mosquitoes,[11] and Eylan and Davidovitch[17] later used the same species in successful transmission experiments in the laboratory. These facts indicate that there may be a transmission cycle involving avian species, mosquitoes, and man; that outbreaks such as those in Israel may be initiated by movement of infected birds from neighbouring endemic areas; and that migratory birds may be responsible for the wide dissemination of the virus through the major continents of the world.

It is the purpose of this paper to show, with the aid of results of protection tests performed on 1,462 human sera and 413 monkey sera, that West Nile virus occurs in the Union of South Africa.

MATERIALS AND METHODS

Reference was made to maps showing population density, vegetation types, altitude, and rainfall. In localities which were inhabited by man and which appeared most likely to provide a reasonably dense population of haematophagous arthropods, human sera were collected. The decision to include the Eastern Cape Province in the first blood-collecting tour was based on the fact that, when testing monkeys to determine if antibody to West Nile virus was already present before inoculating them to produce antibody for experimental use, it was found that 7 of 12 monkeys trapped on a farm in the Bedford area of the Province had naturally-acquired antibody.

In each locality the blood samples were collected from people who were born and had spent their entire life within a few miles of the bleeding place. This limited the majority of the donors to non-Europeans, since one seldom found a European who had not travelled too widely to be included in the collections. At each collection point the donors consisted of approximately equal numbers of children (0-14 years) and adults (15 years and over), and an attempt was made to draw the children from each 5-year age-group.

Our stock virus is put up as frozen or lyophilized 5% or 10% suspension of infected mouse brain in whole, normal, pre-tested monkey serum. The preparations are stored in sealed ampoules in the CO_2 cabinet. Dilutions are made in a 0·75% solution of Armour's fraction-V bovine plasma-albumin in a phosphate buffer. The presence of protein in these solutions is necessary to protect the virus, which would otherwise deteriorate very rapidly.

The results upon which this paper is based are derived from neutralization tests. These are sometimes referred to as protection tests, because a positive result depends upon the presence of antibody in a serum which neutralizes the virus and thereby protects the experimental animals into which the serum-virus mixture is inoculated. In tests with West Nile virus, adult Swiss white mice are the animals normally used. They are anaesthetized and then inoculated intracerebrally with 0·03 ml. of a mixture which contains equal parts of the test serum and approximately 100 times as much virus as would be needed to kill 50% of the animals in each group. The virus potency is checked by a control titration which is carried out with every group of sera tested. Before inoculation the serum-virus mixtures are incubated for 2 hours at 37°C to allow interaction between the virus and any antibody which may be present. The antibody upon which we depend for our results is heat-stable, but it is dependent upon a heat-labile factor in normal serum for a potentiating effect. Therefore, from the moment the serum is drawn from a donor, it has to be handled with care and kept as cold as possible without actually being frozen. As soon as we are able to, we separate the serum from the clot and store it at —20°C. As thawing and re-freezing are deleterious to the complement-like factor, we minimize this by pipetting 0·2 ml. aliquots in fours and storing them at —20°C until required. The specimens must also be bacteriologically sterile, since contamination with pathogenic organisms would give false negative

results, although the illness caused in the mice by bacteria may usually be differentiated clinically. A contaminated serum may be used after filtration through a 'bacteria tight' filter, but the filter may absorb sufficient antibody to mask a weak positive.

All the specimens collected from human beings are taken into sterile vacuum syringes—either 30-ml. vacunes or 20-ml. vacutainers. The blood is allowed to clot at the ambient air temperature. The filled vacuum-syringes are then packed in one-gallon thermos bottles with water ice and kept cold until they are brought to the main laboratory. There they are centrifuged in the cold and the serum is separated, pipetted, and frozen in sealed containers at —20°C.

The monkey sera are all from common vervet monkeys, *Cercopithecus aethiops pygerethrus* F. Cuvier; they were obtained in Johannesburg with the kind permission of Dr. James Gear, Director of the Poliomyelitis Research Laboratories. Whenever practicable, the monkeys received at these laboratories are bled within a day or so of their arrival so that any antibody they have can be related to the part of the country from which they have come. We are able to ascertain the source of each monkey, within a few miles, from all areas except the one referred to as Nylstroom. The monkeys from there are trapped over several miles of country stretching northward from this point, and no record is kept by the trappers of where each monkey was found. The monkeys are anaesthetized with ether or with nembutal and are bled with an ordinary syringe, usually from the femoral vein, although sometimes from the heart.

RESULTS

The 1,462 human sera were drawn from residents of 45 localities situated throughout the Eastern portion of South Africa. These localities, and the number of immune responses obtained from them, are shown in the accompanying map (Fig. 1).

When the results are considered according to provinces, we see that of 353 sera drawn from 13 localities in the Eastern Cape Province, only 4, or 1·1%, were found to have neutralizing antibodies.

In Natal, of 407 sera from 12 localities, 12, or 2·9%, had neutralizing antibodies.

In the Transvaal, of 572 sera from 19 localities, 25, or 4·4%, had neutralizing antibodies.

In the Orange Free State, of 130 sera collected in 4 localities, 27, or 20·8%, had neutralizing antibodies.

Although it appears that West Nile virus does not commonly attack man in the Cape, the fact that it is present in the area is shown by the results for monkey sera. Of 62 monkey sera from Bedford, 23 or 37·1% were positive, and of 127 monkey sera from Cookhouse, which is about 23 miles away, 34, or 26·8%, were positive. Nevertheless, 131 human sera collected in this area were non-protective. What makes these results more striking is the fact that the 41 negative human sera collected at Bedford were all from people living on the farm where the 23 immune monkeys were trapped. One possible explanation for this situation is that the monkeys normally live on a hill that the people on the farm have little or no reason to ascend; therefore, the people do not come into contact with the local vector of the disease.

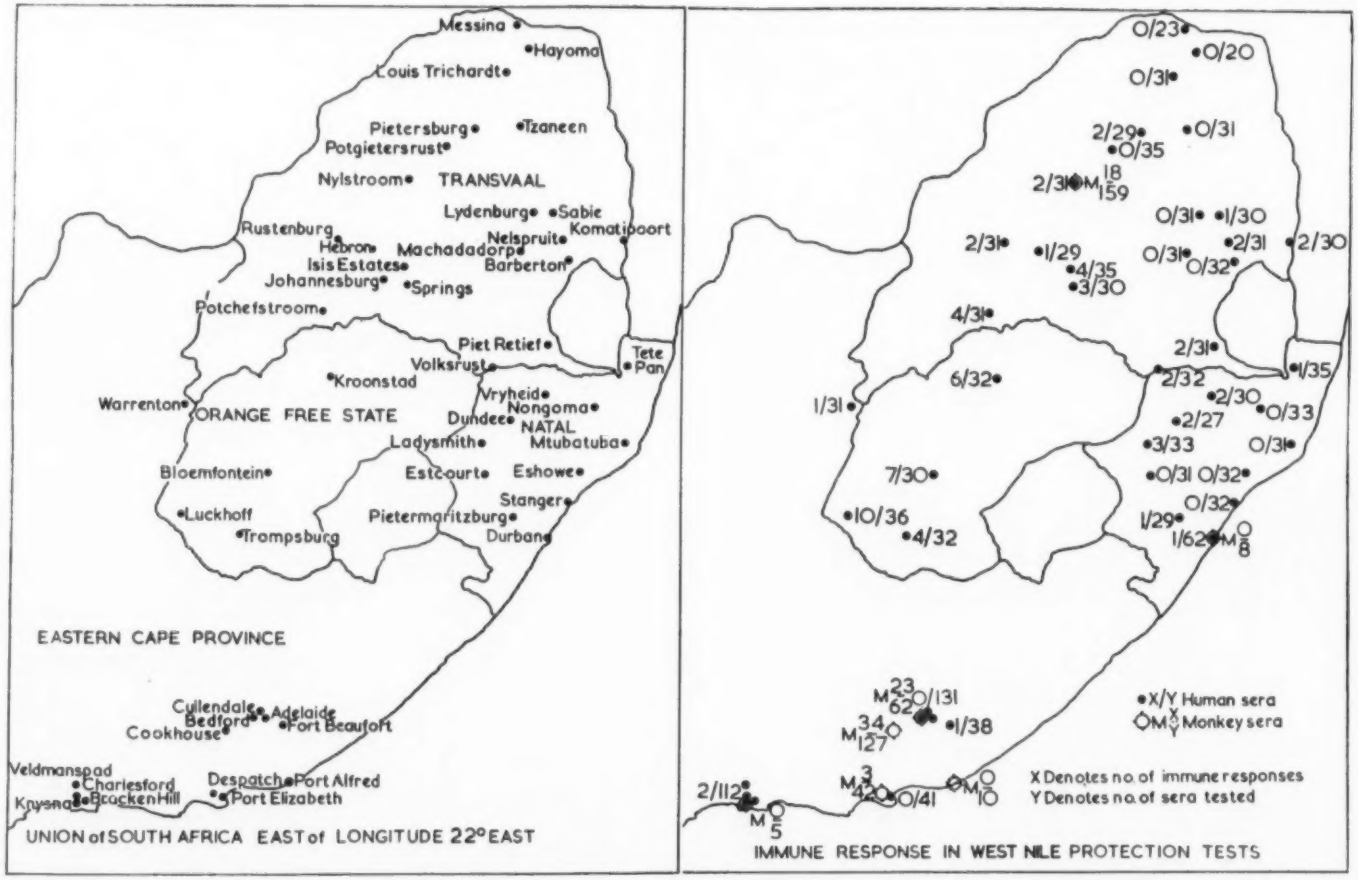
This level of infection for monkeys does not hold throughout the Cape. The results for Despatch show that only 3 out of 42 monkey sera had neutralizing power. Port Alfred and Knysna are represented by 10 and 5 specimens respectively, none of which had antibody. Representing Natal we have but 8 monkey sera from Durban, and these were all devoid of neutralizing power. From the Transvaal we had 159 monkey sera from an area extending north of Nylstroom, and of these, 18, or 11·3%, were protective—not markedly different from the attack rate among the human population of the area. It is unfortunate that we have no monkey sera for the Orange Free State.

Returning to the results with human sera, if we disregard the provincial boundaries and also those localities from which the number of positive results obtained was less than 1 in 25 sera tested, we find that the remaining positive results may be grouped into 6 distinct areas, 5 of which are on the High Veld. What is suggested is not that the altitude is the important factor here; it is that in the areas under discussion, the ecological picture is one which favours the existence of West Nile virus, and also that in these places man comes into contact with the vector of the disease.

The area not situated on the High Veld lies to the east of the Drakensberg, a short distance from the northern boundary of Swaziland, and extends from the mountains to the border of Portuguese East Africa. In this area 91 sera were collected, and of these, 5 (5·5%) gave positive results. We may regard Pietersburg, on the High Veld in the Northern Transvaal, as an area, since, despite the fact that 2 (6·9%) of the 29 sera collected there gave positive results, it is virtually surrounded by completely negative areas. Nylstroom (6·5% positive) is in a similar situation. Five localities in the south-eastern part of the Transvaal and the north-western part of Natal were grouped together and yielded 11 positives (7·2%) of 153 sera. The central and western Witwatersrand gave 14 positives (9·0%) of 156 sera, and 4 localities in the western half of the Orange Free State gave 27 positives (20·8%) of 130 sera. It may be that the last three localities mentioned are part of a continuous area and give the appearance of being separate only because of the gaps between our collection points.

The ages of the donors whose sera gave positive results in the neutralization tests ranged from 4 years to above 90 years, and the average was 37½ years. Little significance can be attached to the sex of the donors since we were unable to obtain equal numbers of males and females at the collection points. We were dependent mainly upon male labourers in some areas and upon housewives in others. In Kroonstad the adults were very old people, their average age being over 80 years. In areas where we had young positive donors, however, we could be certain that the virus had recently been active. For instance, at Isis Estates there must have been virus activity within the last 4 years, and probably less, since our youngest positive donor was 4 years old.

Isis Estates is a well-ordered farm near Babsfontein, about 30 miles N.N.E. of Johannesburg. A large herd of pedigreed Friesland cattle is maintained there for the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Barberton
Isis Estates
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Piet Retief
Tete Pan
Volksrust
Kroonstad
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4/32
1/62
M 8
M 23/62
O/131
1/38
M 34/127
M 3/42
O/41
M 10
2/112
M 5
X/Y Human sera
M X/Y Monkey sera
X Denotes no. of immune responses
Y Denotes no. of sera tested
IMMUNE RESPONSE IN WEST NILE PROTECTION TESTS

Fig. 1

Bader Bros. by a resident veterinary officer, Dr. Ian McFarlane. Our attention was first drawn to the farm by Dr. R. A. Alexander of Onderstepoort, early last year, when there was an outbreak of an illness in cattle which was then undiagnosed. Because further investigations showed that there was illness also among human beings on the farm, it was decided to carry out a programme of work there. It may be noted here that, although we have not, as yet, been successful in isolating West Nile virus from arthropods caught on the farm, we have isolated two strains of another virus from insects trapped there.[18]

Blood was collected from 154 Natives, relatively permanent residents consisting of employees and their families. Of these, 18 showed neutralizing antibodies for West Nile virus. This is somewhat difficult to interpret, since many of these people have spent only a small portion of their life there; 35 donors, however, were born and had spent their whole life within a few miles of the farm, and of these, 4 gave positive results in our protection tests. The immune donors were a 39-year-old female and one of her daughters, aged 4 (her other children, both younger and older than this one, were negative), a 21-year-old male, and a 28-year-old female. There were other donors who could well have acquired their immunity in the area; for instance, one old man over 70, who had been employed on the farm for 30 years and on a neighbouring one for many years before that. However, since he originally came from Nyasaland we cannot be certain that he did not acquire his infection there. The results of a number of tests which were carried out with sera from the cattle on the farm will be reported elsewhere in detail. It suffices here to report that tests carried out on two bleedings, which were collected 10 months apart, show a number of conversions from non-immune to immune status. From this fact we are led to believe that the virus has been active in the Babsfontein area quite recently.

Of 15 wild birds collected on the farm, 5 were positive: 2 black-headed herons, 1 cattle egret, 1 shrike, and 1 sacred ibis.

These results may indicate that birds are involved in natural cycles of infection with this virus.

DISCUSSION

This report covers the greater part of the work done on West Nile virus over a period of 18 months by a team working on arthropod-borne virus diseases. The results of the tests indicate that the virus occurs in a number of areas in the Union of South Africa and attacks man, monkeys, birds, and domestic animals.

No attempt has been made to obtain figures for the attack rate in Johannesburg itself, but it appears to be in an infected area. In this zone, and in the areas in the Free State where we obtained our highest figures, there is a wave of insect activity covering about 3 months of the year—in this case January to March—which is analogous to the situation in Israel. The mosquito, *Culex molestus*, which was incriminated in Israel, does not occur in South Africa, but the very closely related *Culex pipiens* and *fatigans* do occur. Although treatment of the infection is purely symptomatic, active prophylactic measures may be taken by controlling all probable insect vectors.

With regard to transport of the virus, we have demonstrated antibody in the blood of cattle egrets taken on pans about Johannesburg, and specimens of the species ringed in Johannesburg have been recovered as far north as Lake Kivu in the Belgian Congo. Among the other species of wild birds collected at Isis Estates which showed antibody black-headed herons, ringed in this area, have been recovered 100 miles to the north; they appear to move very freely between this point and the Orange Free State. The sacred ibis ringed here are frequently recovered from the eastern portion of Northern Rhodesia. The other species which gave a positive result was the fiscal shrike, which does not have a very great range of movement and presumably is infected locally by the bite of haematophagous arthropods.

A summary was given of 4 reports of the clinical picture induced by West Nile virus in Israel. From these it can be seen that the disease may occur in epidemic form and that it may be of considerable importance. For instance, if an army composed of non-immune troops should be sent to an endemic area during the season of insect activity—for example, Northern Israel during the months of July to September, or the Luckhoff area in the Orange Free State from January to March—it is possible that it could be severely disrupted for a period of some weeks because of infection with this virus. It is suspected that an epidemic which actually did occur among British troops and civilians in Palestine in 1942 was due to this virus.

One may also see that with the various combinations and permutations of the symptoms which it may cause, West Nile virus may induce a clinical picture which is extremely difficult to differentiate from many other conditions, some of the more important ones being dengue, sandfly and tick-bite fevers, infectious mononucleosis, poliomyelitis, and the exanthemata. It is not at all improbable that an illness caused by this virus could be mistaken for measles, rubella, or roseola infantum. One often hears of cases which are spoken of as unusual in that the patient had the extraordinary power of contracting measles more than once. These cases are especially suspect if they occur in an area endemic for West Nile. It is also possible that West Nile virus is the culprit in some cases of P.U.O., whether or not they appear to be responsive to antibiotics.

SUMMARY

Virus neutralization tests with human sera and sera from *Cercopithecus aethiops* monkeys captured in various localities in the Union of South Africa have shown that significant numbers of both human and monkey sera possess humoral antibodies to West Nile virus. The surveys made to date show that the distribution for man and monkeys is not uniform. There are areas in the High Veld where the distribution is most common in human beings. With monkeys it appears that the infection is most prevalent in the environs of Bedford in the Eastern Cape Province.

A summary is given of the reports in the literature

concerned with the clinical aspects of infection with West Nile virus, and the implications of the survey results are discussed.

The collaboration of the Headquarter staff of the Union Health Department and local officials in the various localities, in arranging for the collection of human bloods, is gratefully acknowledged. I am also indebted to Dr. James Gear, Director of the Poliomyelitis Research Foundation Laboratories, for making available many of the facilities required for this work, and to Drs. Kenneth C. Smithburn and Robert H. Kokernot for their advice and collaboration, without which these studies would not have been possible.

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THE OUT-PATIENT MANAGEMENT OF ACUTE LUNG ABSCESS

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Acute lung-abscess is a serious condition. It carries the ever-present danger of the complication of cerebral abscess. Should it not resolve, the chronic state which develops is associated with a miserable and prolonged period of ill health that can only be cured by thoracic surgery.

That an out-patient department may undertake the treatment of this condition may occasion surprise. The Durban Chest Clinic found it necessary to treat certain patients who refuse admission to hospital, usually Natives who are afraid of hospitals, and improvement and cure resulted in certain types of acute lung-abscess thus treated. As practitioners must be faced by the same problem the data of 6 consecutive cases are presented in this report.

The type of lung abscess that responds to out-patient treatment presents the X-ray appearance of a solitary round or oval density, not larger than a tennis ball, and situated in the upper half of the lung fields. A surrounding halo of soft homogeneous densities indicates a perfocal pneumonitis. Usually a fluid level is present. The picture is essentially one of an acute and recent condition, which may be termed a primary pyogenic lung-infection to distinguish it from cavitation and abscess formation secondary to other specific lung conditions such as lung cysts (congenital or hydatid), amoebiasis, malignant growth, pneumokoniosis, actinomycosis, fungal infection and bronchiectasis. These conditions require their specific therapy or surgery and are best managed in hospital. Chronic pyogenic lung-infection, too, presenting radiologically as loculated areas of acute inflammation enmeshed in scar tissue, is not amenable to treatment under out-patient conditions with hope of cure; the associated distortion of bronchi renders the natural drainage of pus inadequate, and lung resection offers the only means of cure.

Yet another cause of lung abscess requiring radical treatment in hospital is bronchial obstruction by intrabronchial foreign body or neoplasm. The history, age of patient and X-ray appearance may give a clue to such obstruction. It should also be suspected when the weekly X-ray check which is undertaken in the management of lung abscess shows unsatisfactory progress, perhaps after an initial clearing. Bronchoscopy is indicated here.

The *clinical picture* of the cases of acute lung-abscess we have seen is characteristic: A middle-aged non-European male presents with a cough of a few weeks duration, has foul-smelling sputum, and shows clubbing of the fingers. Pyorrhoea, dental caries, and a heavy encrustation on the teeth are commonly found. It is believed that episodes of alcoholism would feature if a true history were obtainable. The ages ranged from 29 to 53 years in a series of 11 cases observed. There were no female cases. On the theory that bronchial embolism from aspiration of infected material from the upper respiratory tract causes lung abscess, alcoholism, with its suppression of certain protective respiratory reflexes and association of vomiting, is regarded as the determining factor for this age and sex distribution.

CLINICAL SUMMARIES

Figs. 1, 2 and 3 show in sets the initial, intermediate and end-result X-ray plates, with clinical data, of cases 3 treated as out-patients. Three other cases (not illustrated) present the following treatment summaries:

1. I.R., Native male aged 39 years. Abscess in right lower lobe. Penicillin, 22·6 mega-units over 11 weeks. Sulphadiazine,

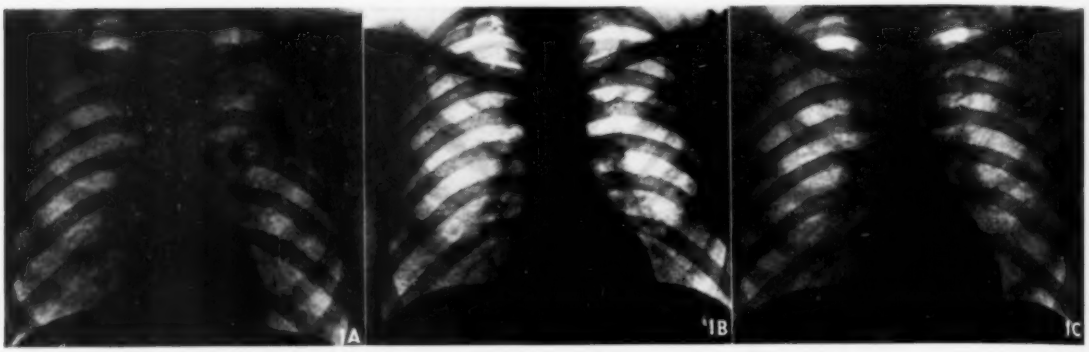

Fig. 1. C.H.P., Indian male, 39 years. Penicillin, 33 mega-units over 11 weeks. Sulphadiazine, 61 g. over 11 days. Potassium iodide, 30 gr. daily over the whole course of treatment. Commenced work as gardener after 18 days of treatment. Symptom-free in 39 days. Faint scar on X-ray in 81 days. No change on X-ray 1 month later.

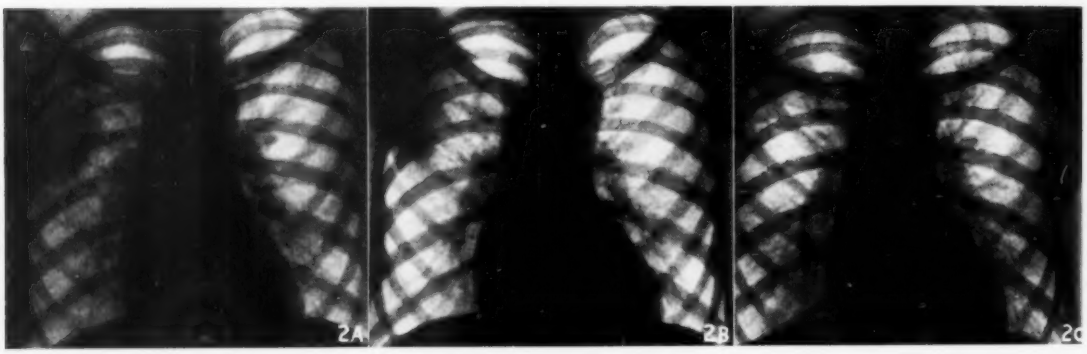

Fig. 2. C.N., Native male, 34 years. Penicillin, 2½ mega-units over 5 days. Sulphadiazine, 48 g. over 9 days. Worked as garden boy from commencement of treatment. Symptom-free in 24 days. Scar on X-ray in 57 days. Follow-up plate a year later showed no change.

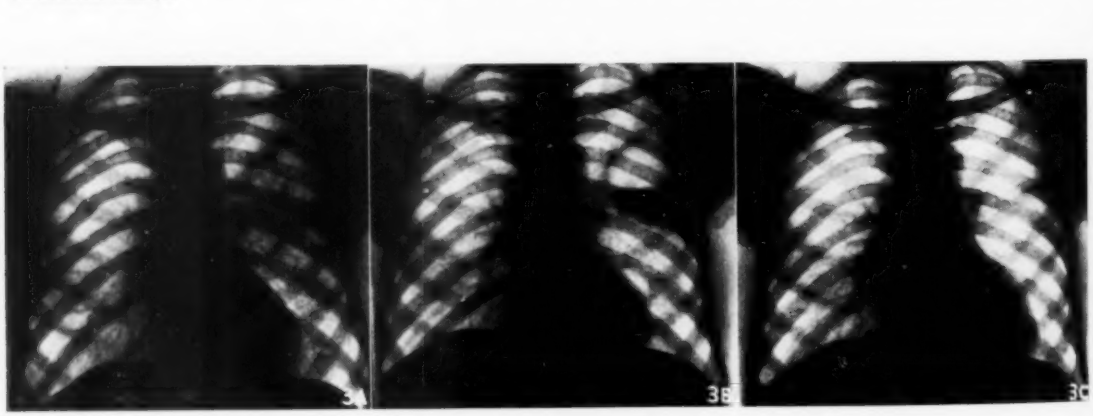

Fig. 3. A.M., Native male, 29 years. Penicillin, 13 mega-units over 4 weeks. Sulphadiazine, 25 g. over 5 days. Potassium iodide, 30-50 gr. daily throughout period of treatment. Worked as builder's assistant from the commencement of treatment. Symptom-free in 21 days. Radiological scar in 28 days.

95 g. over 19 days in interrupted courses. Potassium iodide, 10 gr. *t.i.d.* throughout the period attended. Worked as a garden boy from the commencement of treatment. Sympton-free in 73 days, when X-ray appearance suggested some pathological activity still remaining. Patient could not be persuaded to continue injections of penicillin but continued on potassium iodide alone and cleared up, despite a small relapse, to a faint scar 6 months after commencement of treatment.

2. P.S., Native male aged 53 years. Abscess in left mid-zone. Penicillin, 8·3 mega-units over 3 weeks. Sulphadiazine, 60 g. over 12 days. Potassium iodide, 30 gr. daily over whole period of treatment. Symptom-free and scar on X-ray in 22 days.

3. M.P.C., Indian male aged 33 years. Small abscess in right lower lobe. Penicillin, 2·4 mega-units over 2 weeks. Sulphatriad, 30 g. over 6 days. Radiological clearing in 15 days.

In the 6 cases the average duration of treatment on the penicillin-sulphadiazine-iodide regime was 40 days to 'radiological cure'. Gittens and Mihaly,[1] using penicillin and sulphadiazine on 10 hospital cases of acute lung-abscess, found 67·7 days to be their average for radiological cure. These figures are not offered as a basis for comparison but as an indication of the variation and duration of treatment.

A fairly constant feature of cases treated at this clinic was the disappearance of symptoms in the course of treatment before X-ray appearance suggested pathological inactivity. The gap varied from a few days to a few weeks. It is considered necessary to continue therapy to final radiological cure. Many failures in the medical treatment of lung abscess are due to the premature cessation of treatment when symptoms alone are used as a guide.

SCHEDULE OF TREATMENT

The following schedule of treatment was found to give good results and was used in the cases of this report with occasional variations.

(*a*) Penicillin, 1 mega-unit intramuscularly 3 times a week until cessation of treatment.

(*b*) Sulphadiazine, 2 g. (4 tablets) 3 times a day for 6 days, with a possible repetition of the course.

(*c*) Potassium iodide, 10—15 gr. *t.i.d.* until treatment is stopped.

The schedule has since been modified somewhat. The dose of penicillin has been increased; after an attempt to give 2 mega-units per injection, the dose of 1·3 mega-units was found to be the most practical and convenient at each visit. The preparation used is a combination of the soluble salt and the longer-acting procaine penicillin suspension as found on the market. There have been no untoward effects with the prolonged use of penicillin over months. In view of reports of occasional initial severe allergic reaction to penicillin an initial testing dose of 300,000 units is used.

Sulphamethyldiazine (Sulphamerazine) has been substituted for the parent compound sulphadiazine as being more suited for out-patient use on account of its quicker absorption and slower elimination. The smaller dosage is used of 4 g. *statim* and 1 g. (2 tablets) *t.i.d.* for 14 days.

Potassium iodide is used for the control of yeast and fungal growth which prolonged anti-microbial treatment encourages. Its reputed action of aiding expectoration and dissolving coagulated tissue-exudate would aid bronchial drainage and enhance the penetrating power of the anti-microbial drugs.

Regular X-ray plates, weekly, and later fortnightly, are taken to assess progress and to guide treatment. An initial lateral plate locates the abscess more accurately and gives a clearer definition of its extent. Brock has stressed in his writings the posterior location of lung pyogenic infections as related to dependant drainage during sleep. It has been common in cases at this clinic for the infection to be distributed both anteriorly and posteriorly in the upper or middle lobes. This observation is used in the differential diagnosis from tuberculosis, with its characteristic posterior location. Pulmonary sepsis may present on X-ray as a mottling or infiltration indistinguishable from that of tuberculosis in character. The patient is instructed in postural drainage and is advised to sleep on his sound side.

Should progress as judged by serial X-ray not be satisfactory, there is a strong indication for admission to hospital for further investigation and in order to have facilities at hand for dealing with complications. When considering the continuation of treatment in a tardy case, it must be remembered that a lung abscess requires, on the average, 2 months of treatment to resolve. After the initial few weeks of treatment, resolution as seen on X-ray is usually not spectacular. The possibility has to be considered that a chronic pyogenic lung infection may result if medical treatment is persisted in. Bacteriological tests of sensitivity to the commonly used antibiotics under aerobic and anaerobic conditions are particularly indicated in such tardy cases. The possibility of a Friedlander's infection has to be remembered; this responds to streptomycin and sulphadiazine.

Owing to circumstances, a bacteriological examination of the sputa was not done in all cases. Where it was done, a mixed growth of bacteria was obtained with non-haemolytic streptococci, *N. catarrhalis* and *N. pharyngis* usually in evidence. Oral contamination could not be excluded. It is assumed that mixed infection is present in a bronchial-drained abscess. Where possible, bacterial culture is advised before the commencement of treatment—as also are sensitivity tests. Tubercle bacilli should of course be excluded. The immediate commencement of treatment with the penicillin-sulphonamide-iodide schedule can be made with reasonable confidence before bacteriological data are to hand in any acute uncomplicated lung-abscess of the type described. This procedure would not be out of place in a case in which the abscess was suspected of having a primary cause in the lung, since the resultant clearing of pyogenic infection would bring to light any associated condition and render its investigation easier.

Gittens and Mihaly[1] state that the 'challenging effectiveness of penicillin perhaps relates to the usual pathogens likely to be encountered in an acute lung abscess, i.e. the predominant Gram-positive oropharyngeal flora and the spirochaetal organisms of the oral cavity.' Drake and Mason Somes,[2] in a review of 45 cases of acute lung-abscess, found the following causal organisms:

	Cases
Non-haemolytic streptococci	24
Spirochaetes	12
N. catarrhalis	10
Fusiform bacilli	9
Staphylocci	8
Non-specific Gram-negative bacilli	8
B. pyocyaneus	5
Pneumoccocci	4
Diphtheroids	3
B. influenzae	1

Practically all these organisms are sensitive to penicillin. Gittens and Mihaly[1] compared the effectiveness of chlortetracycline (Aureomycin), oxytetracycline (Terramycin), and a penicillin-sulphadiazine combination, in 37 cases of acute lung-abscess. The average times for radiological cure in the 3 groups were 64·3, 69, and 67·7 days respectively. They conclude that the 'wide-spectrum' antibiotics have no advantage over the penicillin-sulphadiazine combination, and reserve them for cases where the latter fails.

THE ROLE OF SURGERY

There is no uniformity of opinion on the role of surgery in lung abscess. Brock,[3] who has written extensively on lung abscess, regards the approach to this condition as essentially surgical. He considers that as the surgeon deals with accumulated pus elsewhere he should do so in lung abscess as well. While admitting that some cases do clear up on medical treatment alone, he indicates the dangers of persisting with such treatment when response is poor and the greater risks of surgical complications when the cases come to surgery in a chronic state. He compares the results of medical and surgical treatment of 318 cases of lung abscess from 1933 to 1950, a period covering a pioneering phase of thoracic surgery and before the widespread use of chemotherapy. He finds that surgery, with an admittedly high mortality rate of 23% as compared to 7%, yet resulted in a higher rate of cure—65% as compared to 40% after medical treatment. The group surgically treated showed a lower chronicity rate of 10·5% as compared to 52·2%. As no clear distinction between acute and chronic infections is made in this comparison, these findings would be influenced by the application of medical treatment to cases of chronic pyogenic infections, in which it is not effective.

With the development of lung resection and improvement in technique, lower mortality rates in the treatment of lung abscess are found. Brock reviews the work of 6 teams of surgeons from 1944 to 1950 on 272 cases of lung abscess. The average mortality rate was 9·2%; that of the lowest team 3·8%. Over all there was an 82% good result and Brock forsees still lower mortality rates if cases are submitted earlier to lung resection.

Recent workers have reported series of acute lung-abscess resolving on medical treatment alone in almost all their cases. Stivelman and Kavee,[4] in 1949, reported resolution in 19 of 21 cases on penicillin and sulphadiazine. One patient died of a cerebral abscess and relapses occurred in an alcoholic and an epileptic. They stress the need to continue therapy beyond the stage of disappearance of symptoms and conclude that the concept that patients with acute lung-abscess require early surgery needs drastic revision. Gittens and Mihaly[1] in their series of 37 cases of acute uncomplicated lung-abscess (i.e. 'no evidence of chronic suppurative disease or co-existing pulmonary or systemic disease') obtained cure by medical treatment alone in all the cases. They divided the cases between 3 treatment-schedules, viz. a penicillin-sulphadiazine combination, Aureomycin and Terramycin, and found them equally effective. A few cases where response was unsatisfactory in the first group went on to resolution after a change to one of the tetracycline drugs. These cases were dealt with in hospital.

CONCLUSIONS

From the cases treated at this clinic, the conclusion is drawn that certain types of acute uncomplicated lung-abscess can be brought to resolution or minimal fibrosis as out-patients. The good results obtained in the ambulatory treatment of lung tuberculosis, early and moderately advanced, at this clinic suggest that a degree of physical activity as opposed to bed rest is beneficial in chemotherapy. There is probably an optimum degree of physical activity for best results, differing with case and stage of disease. Rest has always been regarded as a cardinal adjuvant to healing in infection. The isolation of the invading micro-organisms by such processes as the organization of coagulated tissue-exudate into fibrous tissue was favoured by rest. Before the advent of antibiotics it might be said that fibrosis was an intended end-result of infection. The present outlook is that pulmonary fibrosis, other than minimal, is not a desired end-result in lung infections. The resultant pleural synthesis involves an appreciable loss of pulmonary function, and the associated bronchial distortion perpetuates a pyogenic infection. Physical activity on the part of the patient hinders the localizing action of typical chronic inflammatory processes and in a sense would favour the spread of the disease process. With antibiotic therapy, however, exercise would have the effect of rendering these drugs more accessable to the pathogens while they are in a freely dividing state. Experimental evidence shows that a culture medium favouring cell division of the tubercle bacillus enhanced the bactericidal effect of streptomycin and isoniazid *in vitro*. It is a well-recognized clinical observation that the anti-tuberculosis drugs show the most spectacular results in cases which on X-ray look active and acute, i.e. where the host's resistance is low. Thus low host-resistance is advantageous in anti-microbial therapy.

Additional factors favouring healing in the series presented are the following: Ambulation acts as a gravitational aid for the natural drainage of pus *via* the bronchi from abscesses in the upper parts of the lungs. These organs are singularly blessed with a system of natural drainage. The increased respiration of moderate physical activity as compared with strict bed rest is considered to have an aspirating action on the pus. The maintenance of good muscular tone in the ambulant patient, as compared to the disuse atrophy of bed rest, must favour healing.

SUMMARY

1. It has been found possible to treat certain types of acute uncomplicated lung-abscess as out-patients and

bring about effective healing. The requirements are situation in the upper halves of the lung fields and moderation in size. Treatment summaries are given of 6 consecutive cases and X-ray illustrations of 3 cases.

2. The out-patient management and the schedule of penicillin, sulphonamide and potassium iodide are outlined.

3. The place of surgery in the treatment of lung-abscess is briefly discussed.

4. The theory is suggested that physical activity on the part of the patient under treatment with antibiotics prevents excessive fibrosis and renders the drugs more accessable to the pathogens. Fibrosis, other than minimal, is an undesirable end-result of modern treatment in lung conditions, particularly pyogenic infection. Reference is made to the beneficial results of treating certain cases of tuberculosis under out-patient conditions on a similar basis. Ambulation aids the drainage of the type of lung-abscess treated by gravitational means.

I wish to thank Dr. B. A. Dormer for encouragement in this work and the Secretary for Health for permission to publish.

REFERENCES

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2. Drake, E. H. and Mason Somes, F. (1951): Ann. Intern. Med., **35,** 1218.
3. Brock, R. C. (1952): *Lung Abscess*, p. 170. Oxford: Blackwell Scientific Publications.
4. Stivelman, B. P. and Kavee, J. (1949): Ann. Intern. Med., **30,** 343.

MINUTES OF THE ANNUAL GENERAL MEETING OF THE MEDICAL ASSOCIATION OF SOUTH AFRICA, HELD AT THE UNIVERSITY OF PRETORIA, ON MONDAY, 17 OCTOBER 1955

The Meeting, which was scheduled for noon, began at 12.15 p.m. as most members present had been unable to reach the meeting place in time. The President (Dr. L. E. Lane, of Port Elizabeth) presided and 69 other members were present.

1. *Notice Convening the Meeting:* The President announced that the notice convening the meeting had been published in the *Journal* of 10 September 1955. It was proposed by Dr. Maister, seconded by Dr. Landau and *Agreed* that the notice be taken as read.

2. *Minutes of Last Annual General Meeting:* The Secretary read the Minutes of the Annual General Meeting held in Pretoria on 28 October 1954. It was proposed by Prof. Davel, seconded by Dr. McMurray and *Agreed* that the Minutes be confirmed. They were then signed by the President.

There were no matters arising out of the Minutes.

3. *Annual Report of Chairman of Federal Council:* The President stated that this Report had been published in the *Journal* of 13 July 1955. Dr. Sichel, Chairman of Council, asked whether there were any questions arising out of the Report. As there were no questions, he moved its adoption, seconded by Dr. J. P. de Villiers. *Carried.*

4. *Financial Statement and Balance Sheet:* The President stated that the Balance Sheet and Financial Statement had been published in the *Journal* of 30 July 1955. The Honorary Treasurer, Dr. J. S. du Toit, said that before moving the adoption of the Financial Statement he wished to make certain comments. He mentioned certain items in regard to the income and expenditure and stated that the Association had ended the year satisfactorily, having added a sum of over £5,000 to its capital.

He then commented on the state of the finances of the Benevolent Fund, drawing particular attention to the donation of over £1,500 which had been made to the Fund by members of the Southern Transvaal Branch. This amount had been raised by various functions, and the Management Committee greatly appreciated the action of the Southern Transvaal Branch and hoped that other Branches would be inspired by their example.

The Honorary Treasurer also referred to the amounts which had been contributed by individuals 'In Memoriam', 'For Services Rendered' and as donations. He spoke of the benevolent work being carried on by the Fund and said he hoped that its accumulated funds would continue to grow so that more could be done with the interest which accrued.

Questions were invited. Dr. B. Epstein asked certain questions which were answered by the Secretary.

Dr. J. J. van Niekerk proposed that the Management Committee be asked to give consideration to the making of grants in respect of the educational requirements of necessitous children of members. He was seconded by Dr. L. Adler and the proposal was *Carried Nem. Con.*

Other questions were asked and answers given.

The Honorary Treasurer then moved the adoption of the Financial Report, seconded by Dr. Charles Shapiro. This was *Carried.*

6. *Election of Auditors:* It was proposed by the Honorary Treasurer, seconded by Dr. H. O. Hofmeyr and *Resolved* that Messrs. Gurney, Notcutt & Fisher, of Cape Town, be re-appointed auditors for the year 1956 at a remuneration of £200 per annum.

7. *Induction of President:* The retiring President, Dr. L. E. Lane, thanked the members for the honour which had been bestowed on him in his election as President, and said that this was much appreciated by his Branch and himself. There could be no greater honour than the trust of one's colleagues and he would always look back with gratitude on this trust and confidence which had been placed in him. The appreciation of this honour was shared with him by his wife and his Branch.

Dr. Lane then introduced the incoming President, Dr. J. H. Struthers of Pretoria. He spoke of the work and interest of Dr. Struthers in all Association matters and said that the honour which was about to be bestowed on him was one which was richly deserved. He then invested Dr. Struthers with the badge of office of President of the Association. *Acclamation.*

Dr. Struthers said that he greatly appreciated the high honour which had been bestowed on him and that he would endeavour to follow the example set by those who had preceded him in the high office which was now his.

Dr. Sichel thanked Dr. Lane for his service to the Association, stating that he had carried out the duties of his high office with ability and distinction.

8. *Other Business:* Dr. B. Epstein referred to the fact that of all the members of Congress a comparative few were present at the meeting. He suggested that consideration be given to wider advertisement of the Annual General Meeting. The Secretary said that the attention of the Head Office and Journal Committee would be drawn to Dr. Epstein's remarks.

The President then declared the meeting to stand adjourned until 8.15 p.m., when it would be reconvened in the City Hall, Pretoria.

ADJOURNED GENERAL MEETING

The Adjourned General Meeting was held in conjunction with the Opening Ceremony of Congress in the City Hall, Pretoria, on 17 October 1955, at 8.15 p.m.

The platform party entered the hall preceded by the mace-bearer, Dr. F. Ziady. The mace had been presented to the Association by the Northern Transvaal Branch a few days previously.

After the singing of Die Stem and God Save the Queen, the Mayor of Pretoria (Dr. H. Muller) extended a welcome to the members of Congress visiting Pretoria, and he conveyed good wishes on behalf of the citizens of Pretoria for a successful Congress.

Acclamation.

The President, Dr. Struthers, introduced the Honourable the Minister of Health (Mr. T. Naudé) and called on him to address the meeting.

In his speech Mr. Naudé mentioned the work which had been done in regard to research into poliomyelitis and the preparation of a suitable vaccine, and spoke appreciatively of the work of Dr. James Gear and his associates. He also mentioned the recent decision of the South African Medical and Dental Council to allow suitably qualified doctors from overseas to be placed on a special register and to be available for circumscribed practice in the country.

Mr. Naudé spoke of the dangers of habit-forming drugs and called on the doctors to exercise great care in prescribing these drugs.

He referred to the shortage of accommodation in mental hospitals which he said was being investigated departmentally, and he felt sure that recommendations which would be made to the Cabinet would receive sympathetic support.

The campaign against tuberculosis was also mentioned and Mr. Naudé referred to the great strides which had been made in domiciliary treatment of these patients. He said that employers could do much to consolidate the success which had been achieved, by re-employing these people.

In conclusion he wished the Congress every success and then declared the Congress officially open. *Acclamation.*

Presentation of Awards: Awards were then presented by the President, citations being read in each case. The awards were as follows:

(*a*) Posthumous award of the Association's Gold Medal to the late Dr. Karl Bremer, for distinguished service to the medical profession (received by Mrs. Bremer).

(*b*) The Association's Gold Medal to Dr. L. I. Braun for distinguished service to the medical profession.

(*c*) The Association's Bronze Medal to Dr. D. P. Marais, Prof. S. F. Oosthuizen and Prof. L. J. te Groen.

(*d*) Certificate of Emeritus Membership to Dr. J. H. Harvey Pirie.

(*e*) Certificates of Honorary Membership to Dr. R. V. Bird and Miss C. A. Nothard.

In addition the following presentations were made:

Hamilton-Maynard Memorial Medal for 1954 to Dr. A. P. Blignault.

Leipoldt Memorial Medal for 1954 to Dr. I. M. Hurwitz.

Past President's Insignia to Dr. L. E. Lane, of Port Elizabeth.

Mrs. Lane, wife of the immediate Past President, invested Mrs. Struthers with the badge of office of President's Lady.

Presidential Address: Before delivering his Presidential Address, Dr. Struthers thanked the Association for the honour which had been bestowed on him in his election as President. He also thanked all those who had contributed to the organization of the Congress. He expressed appreciation to the Minister of Health for opening the Congress and for his address.

Dr. Struthers then delivered his Presidential Address on the subject of 'Medicine and the Community'.

The Presidential Address was received with *Acclamation.*

At the conclusion of the Opening Ceremony the platform party left the hall and a reception followed, at which the guests were received by the President and Mrs. Struthers.

MENTAL HOSPITAL SERVICE TRANSFERS

The following transfers of medical staff in the Mental Hospital Service have been arranged:

Physician Superintendents as from 1 December 1955:

Dr. T. E. Cheze-Brown, Town Hill Hospital, Pietermaritzburg, transferred on promotion to Valkenberg Hospital, Observatory, Cape.

Dr. D. J. Rossouw, Fort Napier Hospital, transferred on promotion to Oranje Hospital, Bloemfontein.

Dr. D. S. Huskisson, transferred from Valkenberg Hospital to Town Hill Hospital, Pietermaritzburg.

Dr. M. Ginsberg, Oranje Hospital, Bloemfontein, transferred to Fort Napier Hospital, Pietermaritzburg.

Assistant Physician Superintendents as from 1 December 1955:

Dr. H. E. Bernstein, Witrand Institution, Potchefstroom, transferred on promotion to Oranje Hospital, Bloemfontein.

Dr. J. S. du T. de Wet, Tower Hospital, Fort Beaufort, transferred on promotion to Sterkfontein Hospital, Krugersdorp.

Dr. S. Weinberg, Oranje Hospital, Bloemfontein, transferred on promotion to Valkenberg Hospital, Observatory, Cape.

Dr. C. G. A. Simonsz, Valkenberg Hospital, transferred to Witrand Institution, Potchefstroom.

Dr. A. J. van Wyk, Sterkfontein Hospital, Krugersdorp, on transfer to Tower Hospital, Fort Beaufort.

Medical Officers as from 15 December 1955:

Dr. N. v. d. Westhuizen, Weskoppies Hospital, Pretoria, on transfer to Komani Hospital, Queenstown.

Dr. K. H. Field, Komani Hospital, Queenstown, on transfer to Weskoppies Hospital, Pretoria.

ASSOCIATION NEWS : VERENIGINGSNUUS

JAARVERGADERING VAN DIE DISTRIKSGENEESHEREGROEP TE PRETORIA

Die volgende is 'n verslag van Jaarvergadering van die Distriksgeneesheregroep van die Mediese Vereniging van Suid-Afrika, gehou op 17 Oktober 1955 om 9.30 vm. in kamer 17 van die nuwe Lettere-gebou, Universiteit Pretoria.

Teenwoordig. Drs. Du Pré Le Roux en Schiller namens die Departement van Volksgesondheid, dr. D. Serfontein van Heilbron, dr. J. H. E. Schults van Witbank, dr. van Schalkwyk van Queenstown, dr. L. J. Botha van Duiwelskloof, dr. Viviers van Komatipoort, dr. Kriek van Mosselbaai, dr. Louw van Virginia, dr. Spaarwater van Pelgrimsrust, en dr. Troskie van Kroonstad.

Dr. Serfontein heet die teenwoordiges welkom en rig 'n spesiale woord van welkom aan die 2 verteenwoordigers van die Departement van Volksgesondheid. Hy meld dat daar gedurende die jaar geen ledegelde gevorder was nie. Verder kon geen notule voorgelees of finansiële staat voorgelê word nie.

Die vergadering is toe oopgestel vir bespreking van sake rakende D.G.-aangeleenthede.

1. *Chirurgiese Fooie*

Dit was gevoel dat die huidige stelsel van werk in verband met die polisie, gevangeniswese, en staande mag ondoeltreffend is. Daar word voorgestel:

(*a*) dat waar 'n Distriksgeneesheer bykomstige dienste lewer aan polisie of gevangenispersoneel, die dienste gelewer besoldig sal word volgens die goedgekeurde tarief van fooie min 10%.

(*b*) dat waar polisie of gevangeniswese-gevalle deur 'n spesialis behandel moet word, dit slegs op aanbeveling van die Distriksgeneesheer sal geskied.

Dr. Schiller sal reël dat die Voorsitter en dr. Troskie die Kommissaris van Polisie sal ontmoet om hierdie aangeleentheid te bespreek.

2. *Bevallings*

Dit word gevoel dat die fooi van £2 per bevalling ontoereikende besoldiging vir die diens is. Die Sekretaris van Volksgesondheid is dit eens dat die fooi £5 behoort te wees maar slegs vir gevalle

waar die geneesheer self teenwoordig is by minstens 1 stadium van die werklike kraam. Dit sal dan ook van die D.G. verwag word om 'n sertifikaat in dier voege uit te reik.

Aborsies. Daar sal geen ekstra besoldiging wees nie, buiten in uitsonderlike gevalle, in welke geval dan die D.G. die spesiale omstandighede van die geval skriftelik sal aanmeld.

3. *Lykskouings*

Dit is die beleid van die Departement van Volksgesondheid om so min moontlik 'n aparte besoldiging vir aparte dienste toe te ken. Vir afsonderlike dienste word 'n eenheidsfooi toegeken en dan ingeskakel onder die gesamentlike fooi wat die salaris behels. Die eenheidsfooi wat lykskouings betref sal hersien word en die nodige wysigings in besoldiging sal aangebring word.

4. *Medisynetoelae*

Die Departement laat tans 'n formulier van medikamente opstel wat as leidraad sal dien om Distriksgeneeshere in staat te stel om te besluit watter medisynes teruggevorder kan word van die Departement van Volksgesondheid, waar sodanige middels in spesiale gevalle uitgereik word.

5. *Reistoelaes*

Die Departement van Volksgesondheid sien hoegenaamd geen kans op hierdie stadium om enige vermeerdering in toelaes per myl gereis nie toe te staan nie. Dit was selfs voorgestel dat die D.G. van die offisiële vervoer gebruik maak in spesiale omstandighede.

Ampsdraers

Die vergadering is voortgesit met die verkiesing van ampsdraers vir die lopende jaar, as volg: Voorsitter—dr. D. Serfontein (Heilbron). Sekretaris—dr. G. F. C. Troskie (Kroonstad). Lede van Komitee—drs. Alan Lloyd (Dundee), L. J. Botha (Duiwelskloof), P. Spaarwater (Pelgrimsrust), H. J. E. Schults (Witbank), en J. A. du P. Kriek (Mosselbaai).

PASSING EVENTS : IN DIE VERBYGAAN

Union Department of Health Bulletins. Report for the 7 days ended 3 November 1955.

Plague. Cape Province. One (1) Native case, (fatal), in the Uitenhage Divisional Council Area. Diagnosis confirmed by laboratory tests.

Smallpox, Typhus Fever: Nil.

Epidemic Diseases in Other Countries.

Plague: Nil.

Cholera: Nil.

Smallpox in Herat (Afghanistan); Moulmein, Rangoon (Burma).

Typhus Fever: Nil.

Report for the 7 days ended 10 November 1955.

Plague, Smallpox: Nil.

Typhus Fever. Cape Province. One (1) Native case in the Glen Grey district. Diagnosis confirmed by laboratory tests. Two (2) Native cases in the Matatiele district. Diagnosis confirmed by laboratory tests.

Epidemic Diseases in Other Countries.

Plague: Nil.

Cholera in Calcutta (India); Chalna (Pakistan).

Smallpox in Kandahar (Afghanistan); Rangoon (Burma); Ahmedabad, Bombay, Calcutta, Madras, Visakhapatnam (India); Dacca (Pakistan).

Typhus fever in Baghdad (Iraq); Cairo (Egypt).

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4th International Congress on Diseases of the Chest. The American College of Chest Physicians has announced that its 4th International Congress on Diseases of the Chest will be held in Cologne, Germany, on 19-23 August 1956. Chancellor Konrad Adenauer is the Honorary President of the Congress and Dr. Gerhard Domagk, who received the Nobel Prize for his discovery of the sulphonamides, is President. Other officers are Professor H. W. Knipping of Cologne (Vice President), Professor Josef Jacobi of Hamburg (Secretary General), and Professor Joachim Hein of Schleswig-Holstein (Chairman of Congress Committee).

The scientific programme is now being organized, and doctors who wish to present original work in diseases of the chest (heart and lungs) are invited to send outlines of their studies to Dr. Andrew L. Banyai, Chairman, Committee on Scientific Program, American College of Chest Physicians, 112 East Chestnut Street, Chicago 11, Illinois, U.S.A. Doctors who have motion pictures or exhibits dealing with heart and lung disease are also requested to submit complete data to the committee. All requests for places on the scientific programme will be given due consideration.

A cordial invitation is extended to physicians and surgeons throughout the world to attend this congress. Further information may be obtained by writing to the American College of Chest Physicians, 112 East Chestnut Street, Chicago.

* * *

Professor Drennan Presentation Fund. Prof. M. R. Drennan, head of the Department of Anatomy at the University of Cape town for 40 years and doyen of the Medical Faculty, recently announced his intention to retire at the end of the year. All his colleagues and former students will wish to honour him on this occasion for the encouragement and inspiration which he has given to them for so many years. With this in view a Committee has been formed with the object of making a suitable presentation to him, and the Committee appeals to colleagues and old students of Professor Drennan to contribute to a Fund for this purpose.

Cheques should be made payable to The Alliance Building Society, which is acting as banker, for the fund, and posted to the Society at P.O. Box 1528, Cape Town.

* * *

Results of Sporting Events at Medical Congress. Following are the results of the sporting events that were held in conjunction with Medical Congress in Pretoria during October.

Ladies' Bridge Tournament. Winner: Mrs. H. Nelson. Runner-up: Mrs. L. Klein. (Organisers of Tournament: Mrs. F. W. McLachlan and Mrs. T. P. Venning.)

Ladies' Tennis Tournament. Winners: Mrs. H. de Villiers and Mrs. M. Slotow. (Organiser of Tournament: Mrs. N. L. Murray.)

Ladies' Golf Competition for the Border Medical Golf Cup. Winner: Mrs. Robin Ross. Runner-up: Mrs. Evelyn Neser. (Organiser of the Competition: Mrs. N. J. van Druten.)

Men's Bowls Competition for the Casewell-Struthers Trophy. Winners: Drs. Vermooten, Coller and Diesler. (Organiser of the Competition: Dr. J. H. W. Casewell.)

Men's Golf Competition for the Campbell-Watt Trophy. Winner: Dr. W. A. Lombard. Runner-up: Dr. E. Samuel. Consolation Prize: Dr. W. T. Ross. Best Gross: Dr. D. v. d. Spuy. (Organiser of the Competition: Dr. J. F. Dippenaar.)

BOOK REVIEWS : BOEKRESENSIES

SPOT DIAGNOSIS

Spot Diagnosis with Notes on Therapy. Compiled by the Editors of *Medicine Illustrated.* Volume II. Pp. 127 with 100 illustrations. 8s. 6d. London: Harvey and Blythe Ltd. 1955.

Most doctors surely indulge in the furtive glance around the 'bus or train (the London Underground is particularly fruitful) to see what clinical syndromes they can detect. This little book brings them home to you. The 100 pictures are fascinating, and nicely produced. The notes accompanying them are up to date, concise

and orthodox. There is a short section at the back on the treatment of some of the illustrated conditions. Medical, surgical, orthopaedic, dermatological, pathological and electrocardiographic photographs are reproduced. Perhaps it is best treated as a 'quiz'—any number of people can play and the highest score wins.

W.P.U.J.

OCCUPATION DISEASES

The Diseases of Occupations. By Donald Hunter, M.D., F.R.C.P. Pp. 1046 + xv, with illustrations. £5 5s. 0d. London: English Universities Press Ltd. 1955.

Contents: 1. Man and his Work. 2. The Industrial Revolution, 1760-1830. 3. Social Reforms in the Nineteenth Century. 4. Health of the Worker in the Twentieth Century. 5. The Ancient Metals. 6. The Other Metals. 7. The Newer Metals. 8. The Aromatic Carbon Compounds. 9. The Aliphatic Carbon Compounds. 10. Noxious Gases. 11. Occupational Diseases due to Infections. 12. Occupational Diseases of the Skin and Occupational Cancer. 13. Diseases due to Physical Agents. 14. The Pneumoconioses. 15. Accidents.

This monumental personal 'Arbeid' by the well-known physician, clinical teacher and examiner is the fruit of a life-long hobby which has permeated almost all the time of this remarkable clinician. He modestly describes it as the experience of 20 years' teaching but the reviewer personally knows that many of the facts and much of the interest which underlie the book permeated the author's clinical teaching nearly 30 years ago. Much of the material has been collected in many parts of the world over those years in breathless so-called holidays. His membership of the British Pharmacopeia Committee has also left its stamp on the book as doubtless on the Pharmacopeia itself. Dedicated 'with affection, gratitude and deep respect' to that great master of exact morbid anatomy and histology, Hubert M. Turnbull, F.R.S., it betrays at least one of the sources of the indefatigable search for information. Only the tribute to his remarkable medical life remains to round off this great achievement. Those who know him will wonder when the time was found even for the dictation of the book.

It is planned and executed with a historical and philosophic 'wholeness' which has almost been lost in the modern craze for progress, and yet it betrays what the author would probably deny, the spirit of the humane social reformer. Starting with a chapter on man and his work, it delves into history, pre-history, natural history and social history in the spirit of the great social reformers.

From this modest beginning it proceeds to the more ambitious task of cataloguing every human ailment which could be attributed directly or indirectly to man's work. In this task the diseases of occupation are dealt with not only clinically, as befits a clinician, but also with a wealth of fascinating detail about the chemical, physical, industrial and social processes which led to the occupational hazard. The author writes: 'In this book it has been my purpose to review on a broad basis and with emphasis on its clinical aspects the problem of disease in relation to occupation. The point of view is that of the general physician; unhappily I have never held a post as factory doctor. In using the clinical approach, I have had always in mind the need to establish the subject on an academic footing, and I have merely put together something of what is known about occupational diseases in order to lay down a basis upon which the practising doctor may build'. Many generations of candidates for the London M.R.C.P. will recognize in the first sentence of this quotation the foundations of their discomfiture at finding that the scope of clinical medicine was surprisingly wider, at least in relation to occupational diseases, than they had ever imagined.

Those of us who practise in South Africa will probably turn at once to the chapter on the Pneumoconioses. We may perhaps be disappointed to see how comparatively small a part the Witwatersrand gold-mining industry plays in this section. There is nothing unique about the problems of the Witwatersrand gold industry, and we shall find at least the clue to any of its problems in the book. As we pursue the book we shall realise that silicosis is only one of many occupational diseases. The book should be on the shelf of every South African doctor and should be thumbed and marked.

Because the reviewer owes much in stimulus and interest to the author, he will be forgiven for pointing out one great defect in the philosophic sweep of the argument. He is disappointed to find in the index that the word 'nutrition' appears on only one page of the text and the words 'food' and 'diet' do not appear at all. In this country, and in many parts of the world, malnutrition is the greatest and most easily preventable occupational hazard. This, however, is a small matter. There are many books on nutrition, but not so many books, at least of this high quality, on the diseases of occupation.

J.F.B.

CORRESPONDENCE : BRIEWERUBRIEK

SPECIALIST AND CONSULTANT REGISTER

To the Editor: The question of Consultant Register *versus* Specialist Register is exercising the minds of the profession. Hitherto the public have not been consulted, even though they pay the piper—or rather the doctor.

The general practitioners have decided what is in the best interest of the public. They have even decided what is in the best interest of the specialist. *Doc Labourer*[1] in your issue of 5 November 1955 writes, 'A new and prosperous era will dawn for the specialist'.

The final issue will be decided by the public, who will insist on a free choice of doctor, which cannot take place under a Consultant Register. So I personally am not so perturbed as *Junior Specialist*.[2]

Senior Specialist

Johannesburg
9 November 1955

1. *Doc. Labourer* (1955): S. Afr. Med. J., **29**, 1060.
2. *Junior Specialist* (1955): *Ibid.*, **29**, 1020 (22 October).

To the Editor: Doc Labourer[1] in his letter on this subject has not assuaged my fears by one iota.

He states that a new and prosperous era will dawn for the specialist once a consultant register is instituted. I contend that hundreds will be economically ruined, and will be forced off the register. Many of the established senior specialists feel the same, and they are in the best position to judge.

In spite of his statement that the Consultant Register will save the patient a lot of time, trouble and money, I contend that from the public point of view, it would be a retrograde step. There would be no free choice of doctor; diversion of patients and dichotomy would be rife. As for his phrase, 'What is the use of asserting that the patient has the right of free access to the specialist when he is unable to exercise this right in an intelligent manner?'—well really! this beats every other specious argument I have heard on this subject.

It is high time the specialists aroused themselves from their apathy and took active measures to ward off the danger that confronts a large and important section of the medical profession as well as the public at large.

Junior Specialist

Johannesburg
9 November 1955

1. *Doc Labourer* (1955): S. Afr. Med. J., **29**, 1060 (5 November).